The Journal of the American Association of Nurse Anesthetists

IN THIS ISSUE

ANESTHESIA FOR PATIENTS WITH MYASTHENIA GRAVIS
James L. Vanderveen, M.D. 243

ESOPHAGEAL RESECTION IN RELATION TO ANESTHESIA
William J. Gillesby, M.D. 256

CARBON DIOXIDE PROBLEMS IN ANESTHESIA
Robert E. Holzgrafe, M.D. 264

ANESTHESIA PROBLEMS IN PEDIATRIC SURGERY
Earle L. Wrenn, Jr., M.D. 270

RADIATION HAZARDS IN DIAGNOSTIC RADIOLOGY
Matthew W. Elson, M.D. 275

NOTES AND CASE REPORTS 279

LEGISLATION
Emanuel Hayt, LLB. 281

HOSPITAL SAFETY
Harriet L. Aberg, C.R.N.A. 283

ABSTRACTS 285

INSURANCE
John C. Maginnis 288

BOOK REVIEWS 290

CLASSIFIED ADVERTISEMENTS 291

INDEX TO ADVERTISERS 296

INDEX TO VOLUME XXVII 297

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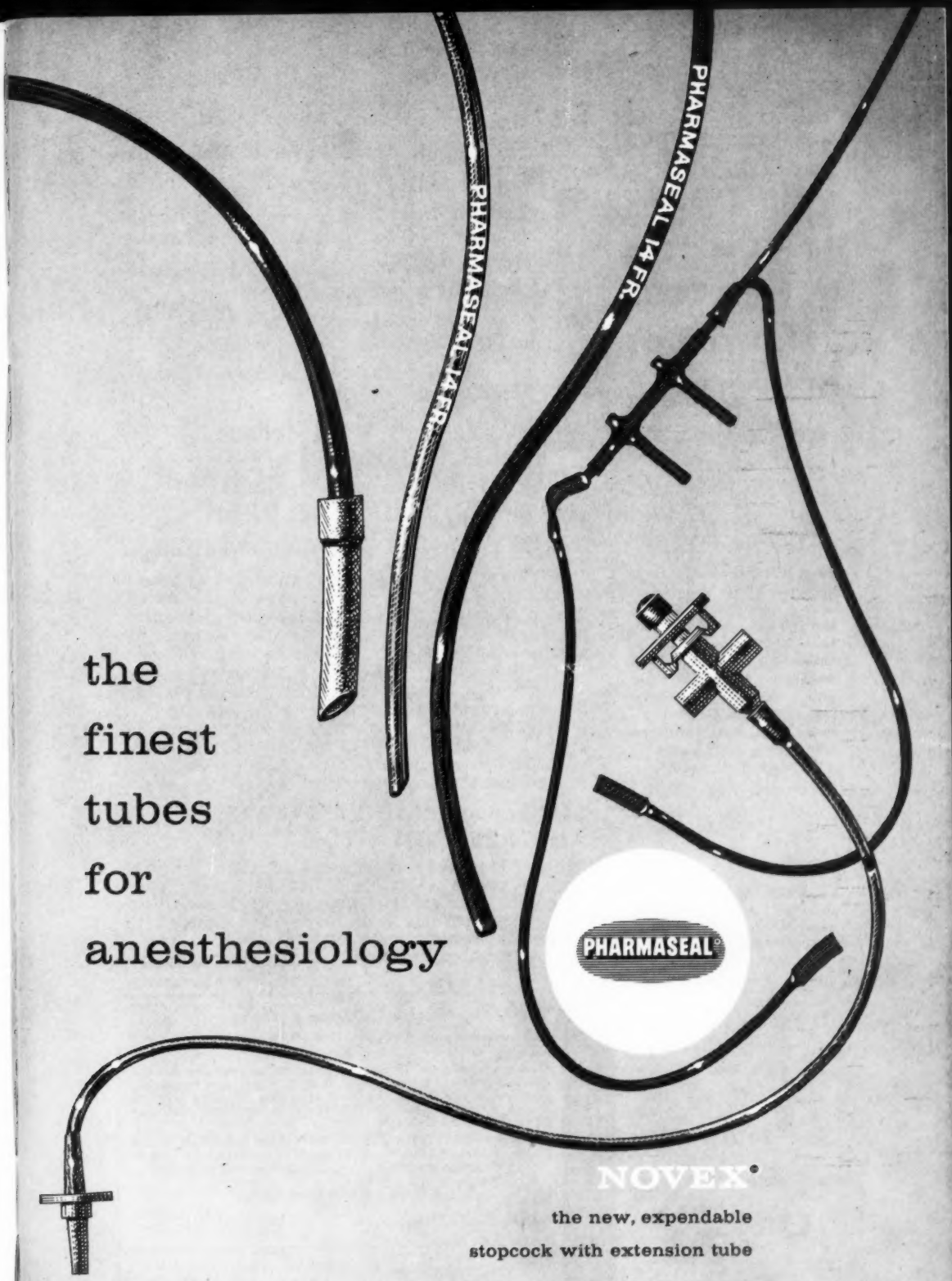

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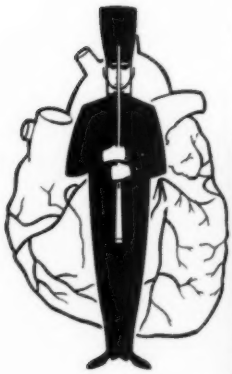

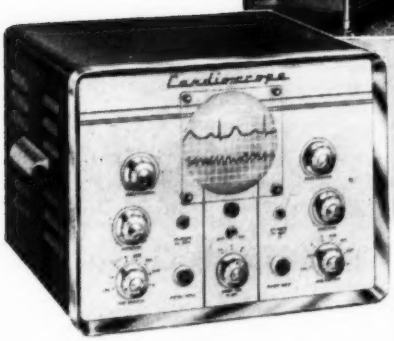

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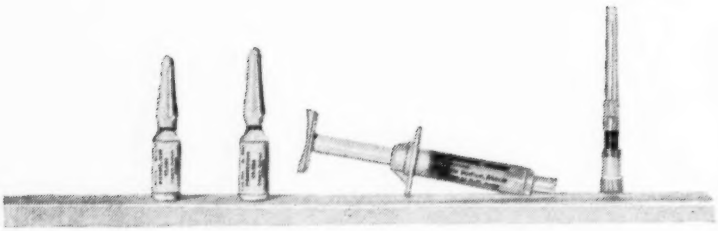

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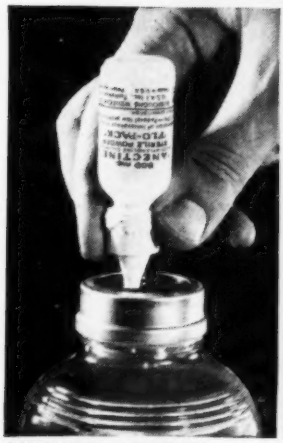

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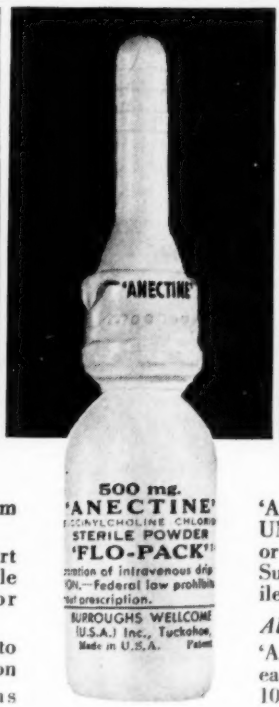

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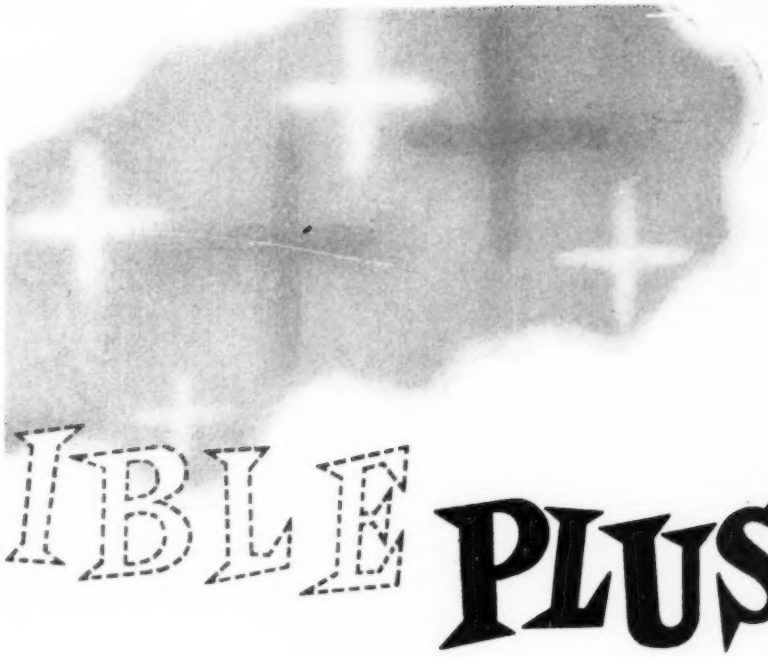

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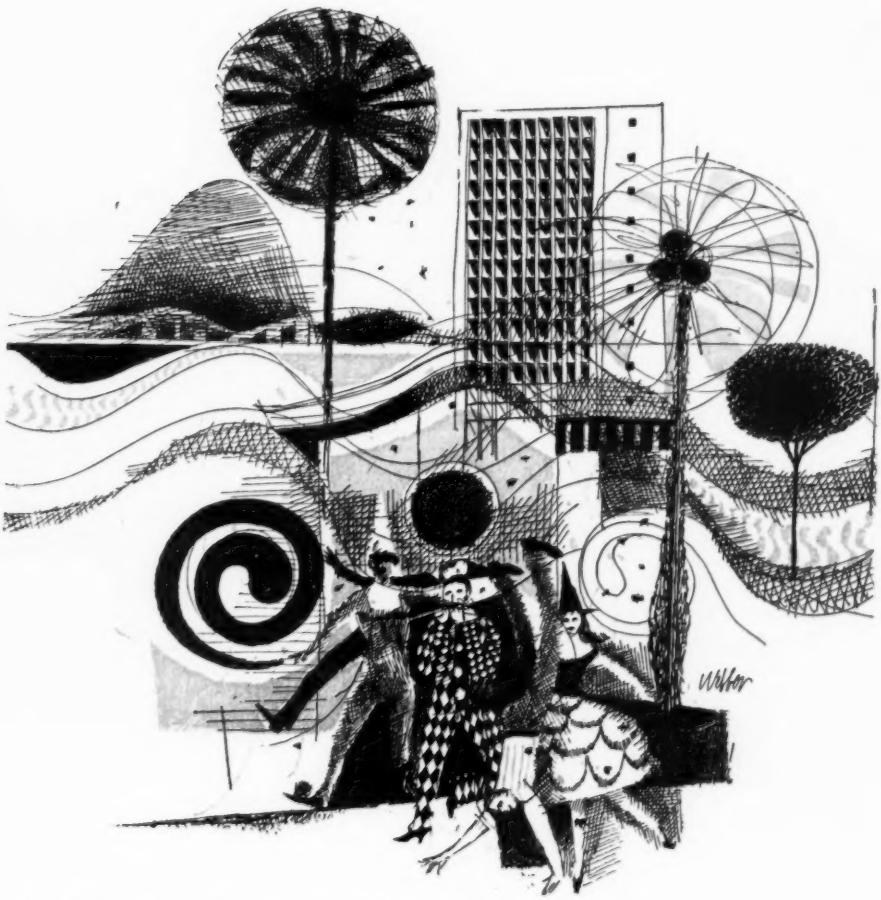

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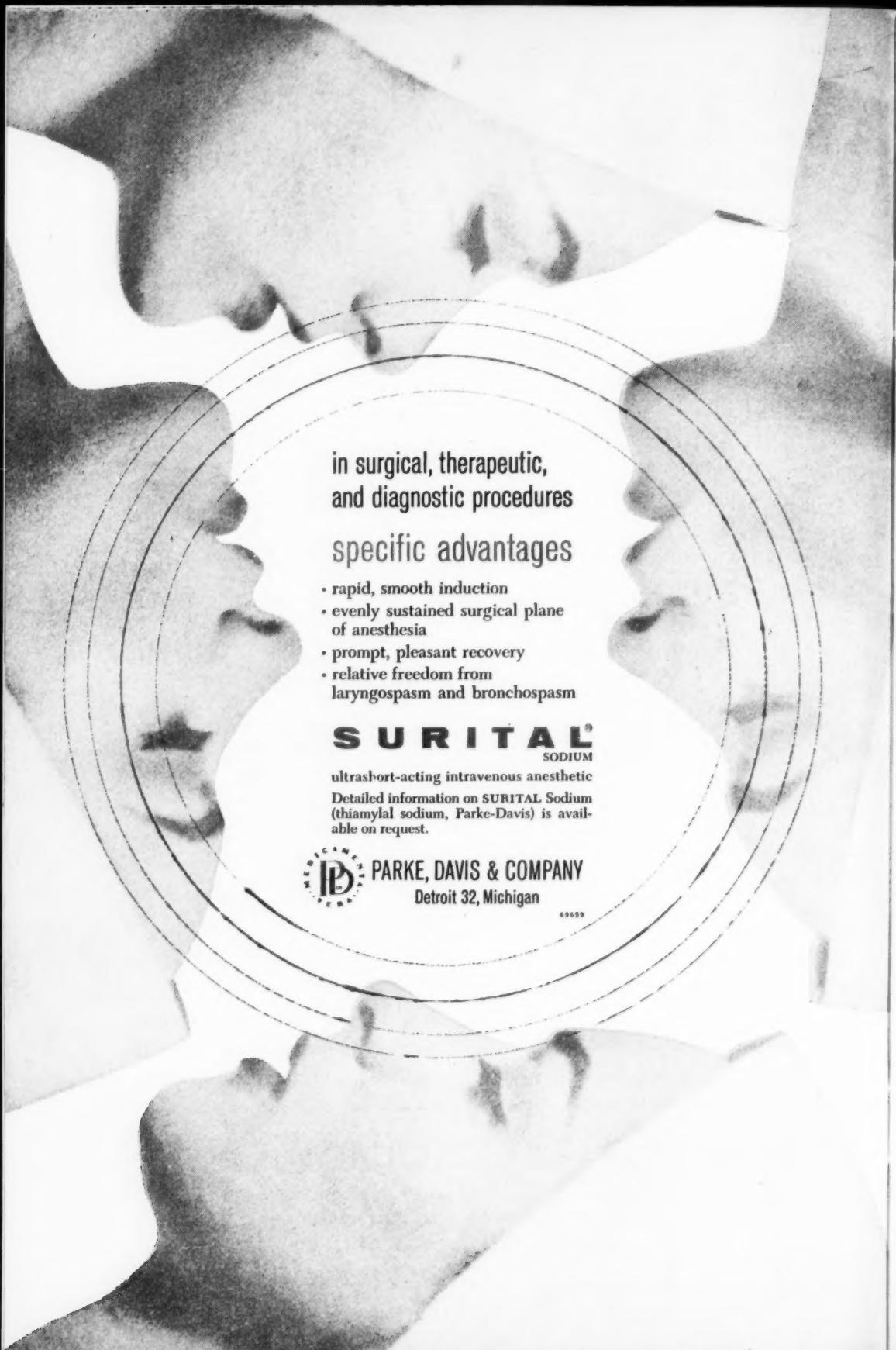
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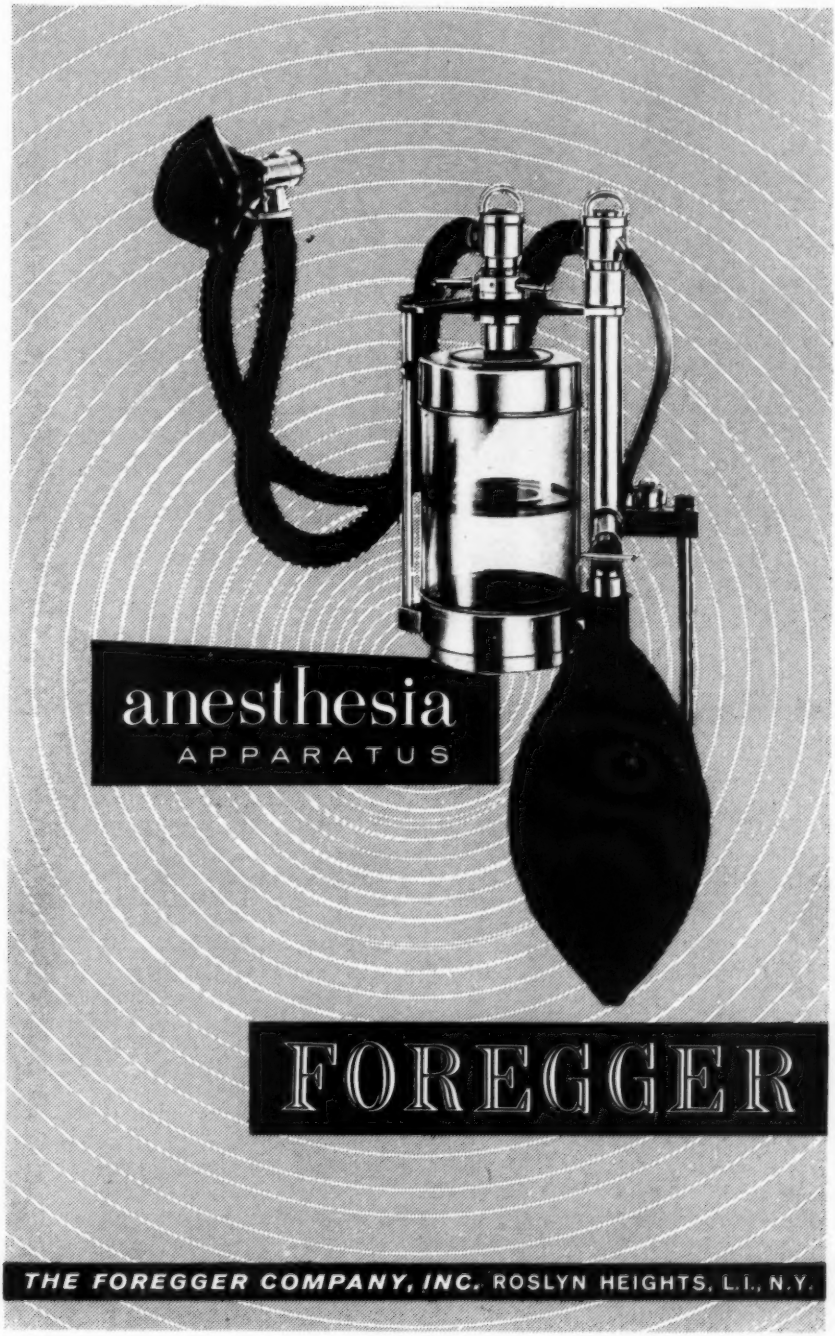
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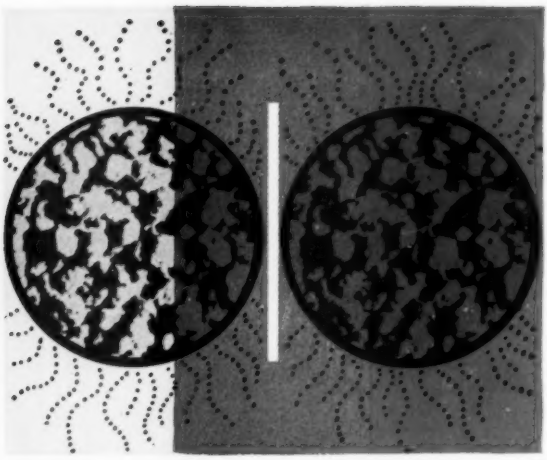

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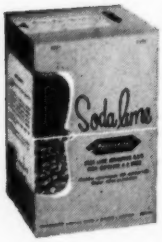

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Anesthesia for Patients with Myasthenia Gravis

James L. Vanderveen, M.D.*
Boston, Massachusetts

Myasthenia gravis has for many years been a disease of particular interest to pharmacologists and anesthetists, since the muscular response of myasthenics and that of the partially curarized patient is strikingly similar. In addition, anesthetists have a special stake in its study because it is the one disease condition in which there is general agreement supported by experimental evidence that one type of anesthesia is better than another.

In this presentation I shall first review the clinical features of the disease, then consider its physiology and the related pharmacology. Finally, I will discuss the problems these patients present when undergoing surgery and anesthesia, with special reference to our experience at the Massachusetts General Hospital and how it has affected our approach to the problem.

Myasthenia gravis was first recognized by Thomas Willis in 1675, and classical descriptions of the disease were written by Erb, in 1879 and Goldflam, in 1893. Jolly, in 1895 designated the disease by its present name.

Myasthenia gravis is a disease of muscles. Most often affected are those of the face, the masseters, the muscles of deglutition and speaking, and the muscles of the eyes and eyelids although it may involve any group of muscles. Easy fatigability of the affected muscles is the basic symptom. Involvement of the respiratory muscles is not uncommon in advanced stages of the disease and is the usual cause of death. It occurs in adults as a rule, but occasionally is seen, even in a profound form, in young people. The cause of myasthenia gravis is unknown. Pathologic changes in the thymus gland, as we shall see later, are a frequent occurrence, but how these are connected to the physiologic changes is not known. The improvement which sometime accompanies pregnancy arouses suspicion of some endocrine defect.

Pathologically, no changes are noted in the nervous system, and there is no atrophy of the involved muscles. Only enlargement of the thymus with lymphoid hyperplasia and the occurrence of microscopic collections of round cells or "lymphorrhages" in skeletal muscles serve to distinguish the tissues of the myasthenic from those of the normal individual.

Clinically, the chief complaint is rapid fatigability of certain muscles,

*Research Fellow in Anesthesia, Massachusetts General Hospital, Boston.
Presented at the annual meeting, New England Assembly of Nurse Anesthetists, Boston, March 23, 1959.

brought on by repetitive effort. In attempting to comb the hair, for example, it becomes impossible to raise the arm above the head more than once or twice. Reading becomes impossible because rapid movements of the eye muscles are affected, and diplopia may result. Some individuals also cannot chew without the rapid onset of fatigue in the jaws. Speech becomes an effort, and the voice may undergo alteration of tone; swallowing is difficult. Ptosis and inability to close the eyelids tightly is common. Sudden attacks of dyspnea and collapse are frequent, and often fatal, but the disease may run a subacute or chronic course for many years, and may even, in some cases, disappear temporarily or permanently.

When tracings of muscular effort are made, either of spontaneously initiated movements, or those induced by repeated electrical stimulation, a characteristic pattern is displayed. The muscular contractions become weaker with each stimulus and soon disappear entirely. With a short period of rest the muscle regains normal excitability and the process may then be repeated.

The pathologic physiology of the disease is confined to the neuromuscular junction. Theoretically there may exist three situations, each an adequate explanation for the pathogenesis of the disease: (1) A deficiency in the amount of acetylcholine produced at the motor end-plate, as might result from a decreased ability to synthesize or release the ester; (2) The existence of some abnormal substance or metabolite with a curare-like action which elevates the threshold of skeletal muscle to the acetylcholine released by motor nerve impulses; or (3) An increase in muscle cholinesterase. The last named possibility has been rather well ruled out by direct measurements of cholinesterase levels, but which of the two remaining possibilities is involved is not known. Regardless of the exact etiology of the disorder, the anticholinesterase drugs afford symptomatic relief and improve muscular strength in part, by increasing the effective concentration of acetylcholine at the myoneural junction.

There are many similarities between the myasthenic muscle and the partially curarized muscle, and the extreme sensitivity of the patient with myasthenia gravis to curare is well known. Curare, like quinine, has been employed as a diagnostic agent to accentuate the muscular weakness in patients suspected of having the disorder but not exhibiting objective evidence at the time of the examination. One-twentieth to one-tenth of the adult curarizing dose is given intravenously; this dose of curare is insufficient to cause significant paresis in the non-myasthenic but quickly evokes marked signs and symptoms in individuals with the disease. The test is terminated within two or three minutes by the intravenous injection of 1 mg. of neostigmine combined with 0.6 mg. of atropine. Neither curare nor quinine should be used unless the result with neostigmine or edrophonium alone is indecisive.

Although myasthenia gravis was first mentioned as an entity in 1675, adequate therapy was not proposed until 1895 when Jolly suggested that physostigmine might afford relief. This suggestion was not acted upon until 1934 when Mary Walker, im-

pressed by the similarity between myasthenia gravis and the symptoms produced by curare poisoning, employed physostigmine with beneficial effect, on the basis of its known decurarizing action. The following year she reported that the use of neostigmine afforded better clinical results. Since that time numerous publications have confirmed the fact that neostigmine is the drug of choice in the therapy of this disease.

The dose of neostigmine varies with the severity of the disease, the response of the patient and such emergencies as difficulty in breathing or swallowing. Whenever possible the patient is placed on adequate oral therapy. The onset of effect is slower with oral than parenteral therapy, but the persistence of action is greater, and the intensity of action more uniform. The total daily oral dose varies from as little as 15 mg. to as much as 375 mg. The interval between doses is of paramount importance; the best dosage schedule must be ascertained by trial and error for each patient and changes made as the need arises. Frequently therapy is required day and night. Larger portions of the daily dose may be given at times of greatest fatigue, such as in the afternoon, or just prior to meals.

Parenteral therapy is reserved for more severe cases. However, a number of ambulatory patients on oral neostigmine may also require appropriately spaced subcutaneous injections. Parenteral injection may be life saving in emergencies, for example during an intercurrent infection.

Inasmuch as myasthenia gravis is the only disease characterized by muscle weakness which is dramatically improved by neostigmine, its subcutaneous or intravenous administration is used as a diagnostic test for the disease, and because it carries little risk, is preferable to curare for this purpose.

Pharmacology

I. Anesthetics

Ideally an anesthetic for use in myasthenics should have the following properties:

1. Non-irritating to the airway (as secretions are frequently a problem)

2. Potent (so that muscle relaxants need not be used)

3. Lack of curariform effect

4. Freedom from dangerous side effects (these patients tolerate additional stress poorly)

5. Rapid elimination (to minimize postoperative respiratory depression)

6. A low incidence of postoperative nausea and vomiting (so that oral prostigmine may quickly be resumed)

a. Di-ethyl ether, the most venerable of anesthetics, and one still unsurpassed in utility and safety, gains a portion of its ability to produce profound muscular relaxation from a peripheral effect at the myoneural junction, and this seems to mimic the action of curare. For this reason one might expect it to perform poorly in this disease, and clinical trial has proved this to be true. In addition it is irritating to the respiratory mucosa. The recovery period may be prolonged, and nausea and vomiting postoperatively may be prominent.

Other ethers we must assume possess similar pharmacologic actions at the myoneural junction, and therefore also to be poor choices, although we have had no experience with them in this group of patients.

b. **Thiopental sodium** (Pentothal® Sodium) and similar short-acting barbiturates possess no curariform properties, so would not seem contraindicated on this most important basis. They are nonirritant, and properly managed, produce a speedy recovery with little metabolic disturbance. However, it is impossible to produce the required depth of anesthesia safely, and for this reason we have not found them useful in thymectomy. The barbiturates might have some importance in operative procedures requiring only light anesthesia, but the well known respiratory depressant effects of these agents would seem to indicate their employment with great caution and in very small doses.

c. **Nitrous oxide** (and Ethylene) is nonirritant, free from effect at the myoneural junction, is perfectly safe from the standpoint of side effects, and produces a rapid recovery. If it were more potent, in this situation, as in anesthesia as a whole, it would be ideal, but a depth of anesthesia sufficient for thymectomy cannot be obtained by its use. There is no reason not to sanction the use of either of these agents in myasthenics, when the requirements of the surgery make the light level of anesthesia obtained sufficient.

d. **Cyclopropane,** the remaining general anesthetic in common use, is nonirritant, and the dangerous side effect of cardiac arrhythmia is not often seen at the level of anesthesia required for thymectomy. It is potent enough to be used alone for surgical procedures of this magnitude, being able to suppress the reflexes initiated by dissection in the mediastinum. It gains its muscular relaxing ability from a mode of action in which there is little, if any involvement of the myoneural junction. It has much to recommend it. In addition, being rather insoluble in body tissues, the recovery time is brief, spontaneous ventilatory effort is rapidly regained, and the incidence of postoperative nausea and vomiting is low.

Cyclopropane has been the most useful general anesthetic agent in our hands for this group of patients, and is the one which we believe, without question, to be the agent of choice for thymectomy.

e. **Local anesthetics** are said to have a curariform activity as one of their side effects. Experience has shown that in the clinical dosage range this is insignificant in both the normal and the myasthenic. Operations capable of being done under local anesthesia may be safely performed in this manner on myasthenics.

These same agents may be properly used for regional or spinal anesthesia, and have been so employed in the presence of myasthenia gravis.

f. **Topical anesthetics** having the same basic chemical structure as, and being often identical with, those used for local, regional or spinal anesthesia, carry no more hazard in general, and often less, since they are used in smaller total dosage.

II. Muscle Relaxants

a. "**Curare**" (d-tubocurarine [Tubocurarine®] and d-chondrocurarine [Intocostrin®]) has been incriminated above as a hazardous drug in the myasthenic. Because of the physiologic deficit which exists in the area of action of curare (i.e.,

the receptor area) the concentration of acetylcholine necessary to break through the curare block at the end plate receptors is much more slowly reached than in the normal individual, or an adequate level may never be attained. Even with the assistance of neostigmine or other cholinesterase inhibitors, this may still be true. If such is the case, recovery from curarizing effects in the myasthenic may not occur until the curare has been redistributed and metabolized. This might require hours or even days.

All our physiologic and pharmacologic knowledge weighs strongly against the use of curare and similar compounds in patients suffering from myasthenia gravis because of the prolonged and unpredictable activity which they may be expected to exhibit.

b. **Gallamine triethiodide** (Flaxedil®), **dimethyl Tubocurarine** (Metubine®), and **benzoquinonium** (Mytolon®) are similar in structure and mode of action to curare and ought similarly to be impugned.

c. **Decamethonium** (Syncurine®) the prototype of the depolarizing agents, and

d. **Succinylcholine** (Anectine®) by their mechanisms of action as outlined previously would seem to exert their effect at the myoneural junction in a physiologic area which in the myasthenic is presumed to be normal. Therefore the use of these relaxants in such cases would seem safe, and indeed, reports of the successful use of depolarizing agents in the presence of myasthenia gravis exist.

We have been reluctant to try these drugs, however, because of a poorly understood phenomenon which is occasionally observed in normal patients. This consists of a persistence of muscular paralysis well past the expected duration, in some cases lasting many hours. It has been suggested that in certain situations these drugs may play a dual role, initially acting by depolarization, and later, perhaps at an intermediate stage in their hydrolysis or after large doses, acting as competitive or curare-like blockers. Recent evidence indicates that this duality of action formerly regarded as a rare occurrence may be quite common, though not often clinically evident. Should a profound prolongation of action take place during administration to a myasthenic, disaster could easily supervene. We would strongly advise against the use of depolarizing muscle relaxants until their pharmacologic action is more clearly understood.

III. Supplemental Drugs

In this class are placed all the many compounds other than anesthetics and muscle relaxants which may be employed in the course of an anesthetic administration. Usually they are given for a specific effect (e.g. the opiates for analgesia, vasopressors to correct hypotension, etc.) The primary effect is as useful in a myasthenic as in a normal patient, and at times as necessary. It is the side effects of which one must be wary. Some of the outstanding examples of commonly used drugs possessing dangerous attributes are listed below. Many more might be added as recent advances add increasing numbers of useful drugs to the anesthetist's armamentarium. This makes it incumbent upon any person dealing with this type of patient to understand fully the pathologic physiology of the disease, and all the pharmacological properties of any drug he in-

tends to employ, and to weigh the possible harm a given drug may do against the good he seeks to accomplish.

a. **Barbiturates** of the moderately long and long acting types are commonly employed as preoperative medication. All barbiturates are respiratory depressants and this feature has caused us to avoid their use when possible and to keep the dosage to an absolute minimum in those patients whose extreme apprehension makes some such euphoriant seem necessary.

b. **Opiates** are even more potent respiratory depressants than the barbiturates. We avoid them entirely as premedicants and confine their use as postoperative analgesics to the minimum and then at a reduced dosage level.

c. **Parasympatholytic drugs** are useful because of the stimulation of the parasympathetic system occasioned by the neostigmine these patients routinely take. It is necessary for many myasthenics to combine oral or parenteral atropine with their maintenance anticholinesterase medication to counteract its visceral effects. By the same token, failure to give adequate doses of atropine preoperatively may lead to great difficulty from secretions during anesthesia. In some patients the preoperative atropine may need to be given in greater doses than one would give to a normal individual, and it is certainly proper to give additional intramuscular or intravenous doses during the course of anesthesia if secretions are troublesome. Repeated administration of atropine may also be necessary if marked bradycardia develops during surgery as a result of the combined vegal stimulating action of neostigmine and cyclopropane.

d. **Quinidine** is the optical isomer of quinine. The curare-like action of this latter compound is well documented. Not so often appreciated is the fact that quinidine may have a similar effect. Care is necessary in its administration to myasthenics. It is important to enquire if any known sensitivity to quinine or quinidine exists.

Thymectomy in the treatment of myasthenia gravis is an operation now more than twenty years old. The benefit achieved by removal of the thymus varies widely. Some clinics abandoned the operation because the mortality rate was so high and the number of patients improved so small. We have persisted because by attention to detail we have been successful in reducing the mortality rate from 40 per cent to under 4 per cent. At the same time rigid selection of patients for operation has increased our percentage of patients experiencing a complete remission or vast improvement to more than 60 per cent. This contrasts with a spontaneous remission or cure rate of under 15 per cent.

Individuals with myasthenia gravis occasionally require anesthesia and surgery for other conditions, just as does the non-myasthenic person. Our experience with anesthesia for thymectomy can be used as a starting point in planning the management of other surgical procedures in patients with this disease.

During the past 19 years nearly 160 myasthenics have undergone thymectomy at the Massachusetts General Hospital. This is, to my

knowledge, the second largest series in the world and is the largest carefully prepared and followed series. As a result of our experience with this group of patients considerable information bearing on the successful preparation and postoperative care of myasthenics has been accumulated, and a very definite and successful technique of anesthetic and surgical management has been evolved.

Preoperative Preparation

Since myasthenic patients present such a special problem in preoperative preparation, it has been our practice to bring them into the hospital well in advance of the anticipated time of operation for careful evaluation of their general physical status, a precaution which we believe is of paramount importance in their successful management. Every effort is made to bring these patients to surgery under optimal conditions, for they tolerate medical and surgical complications exceedingly poorly. Of special importance is the correction of pulmonary infection if it exists. These patients need all the respiratory reserve which only a normal pulmonary system can provide postoperatively. They will not tolerate any interference with their ventilatory mechanism, beyond that imposed by their basic disease and the effects of surgery. The respiratory system should be a particular concern of the anesthetist in his preoperative assessment, and any indication of active pulmonary infection, or of impaired respiratory reserve should be fully investigated and treated.

By the same token the oxygen carrying capacity of the blood is vitally important, and the correction of anemia is mandatory.

Infection in any part of the body ought not to be overlooked. If a well-controlled myasthenic can be thrown into crisis by a minor infection, how much more dangerous must it be to have such an event superimposed upon the stress of operation?

A second reason for the longer period of preparation required of myasthenics prior to surgery is to allow for the change from the usual oral route of anticholinesterase medication to the parenteral route. Although a rough guide has been established whereby the intramuscular dose is found to equal 1/30 of the oral dose, this may vary somewhat from one individual to another, and may require several days of observation, and adjustment b e f o r e a satisfactory schema can be established. When the patient's strength is normally maintained by the proper dosage schedule through this new route of administration, it is safe to change to the intravenous route on the day of operation, the intramuscular and intravenous doses being equivalent.

Anesthetic Management

It is preferable to use local, regional or spinal anesthesia for procedures in which the demands of the surgery make this possible. Caution must be employed in the use of spinal anesthesia; a spinal level high enough to affect the respiratory muscles even in small degree, may produce hypoventilation.

The technique outlined below is that used in our department for patients undergoing thymectomy. It is a satisfactory technique for use during any operation on a myasthenic requiring general anesthesia.

1. Premedication

Atropine alone is the usual pre-

medication. The dose for an average patient is 0.6 mg. or more. Because of profuse secretions which are common when neostigmine is taken the administration of a parasympatholytic agent is even more important than is normally the case. The dose can be tailored on the basis of the patient's prior experience with excessive secretions. There should be no hesitation in giving additional atropine prior to or during anesthesia if secretions are not adequately suppressed. Profuse secretions often interfere with a smooth induction of anesthesia by initiating coughing or laryngospasm.

Opiates and barbiturates are avoided as premedicants. Their respiratory depressant effect may prolong the induction of anesthesia. In conjunction with a depressant anesthetic agent, splinting of the chest due to wound pain, or difficulties in neostigmine dosage regulation, serious ventilatory problems may be produced in the postoperative period.

Only those few patients whose extreme apprehension make some euphoriant seem highly indicated receive any drug other than atropine. A moderately long acting barbiturate (such as pentobarbital) is then given, but in decreased amount.

The premedication is given 45 minutes to one hour prior to the induction of anesthesia.

2. Neostigmine Infusion

An intravenous neostigmine infusion is begun in the patient's room 1½ to 2 hours prior to surgery. One-third to 1/2 of the calculated daily parenteral dose is placed in an equivalent fraction of the daily fluid intake. This is regulated so as to run in at a rate which will just bring about its completion in the required time (i.e. daily parenteral dose = 12 mg. Four mg. are placed in 1000 c.c. of intravenous solution, usually 5 per cent dextrose in water, and regulated to run at 125 c.c./hr. or 33 drops/min. The entire solution would be infused in 8 hours, thus providing 1/3 of the calculated neostigmine and 1/3 of the average daily fluid requirement in 1/3 of the day).

The rate at which the infusion is to run is clearly marked on the bottle and those caring for the patient are carefully instructed to frequently check the rate of infusion.

3. Induction of Anesthesia

Anesthetic induction is performed with cyclopropane and oxygen, usually a 50 per cent mixture. The anesthesia is deepened until sufficient muscle relaxation and depression of laryngeal reflexes has occurred to permit direct laryngoscopy.

Coughing, breath holding and laryngospasm are not infrequently encountered. They are most often due to secretions in the airway, which should be dealt with by suctioning and the intravenous injection of additional atropine.

4. Topical Anesthesia

The larynx is exposed and thoroughly sprayed, together with the trachea, using a topical anesthetic. We have used cocaine 4 per cent and Xylocaine® 2 per cent with epinephrine 1:100,000 and have found both satisfactory.

This maneuver permits the depth of anesthesia to be less than would otherwise be the case when an endotracheal tube is employed.

5. Endotracheal Intubation

If sufficient depth of anesthesia still exists after spraying of the airway, an endotracheal tube is inserted.

If not, the face mask is reapplied and anesthesia again induced to the same level as before, so that intubation can be performed without trauma or inciting noxious reflexes.

We have used both uncuffed tubes under a face mask and cuffed tubes attached directly to the anesthetic machine. Our preference today is for the latter, as this makes the attainment of a tightly closed system easier. The possibility of inflicting trauma upon the tracheal mucosa with an inflated cuff seems small if the pressure used is only just sufficient to produce a seal around the tube. The duration of this operation is not usually long. Should it become so, occasional brief deflation of the cuff to permit temporary resumption of normal blood flow through the capillaries of the mucous membrane in contact with the cuff seems wise. Our practice has been to deflate the cuff five minutes of every hour.

The use of an uncuffed endotracheal tube under a face mask presents two problems. One is the trouble sometimes experienced in obtaining a sufficiently tight closure around the edges of the mask to permit the use of a closed system and assistance to respiration. The other is the occasional difficulty with inflation of the stomach when gases are forced down the esophagus during positive pressure assistance to respiration.

The use of an endotracheal tube is essential because it assures an ever patent airway in these patients in whom adequate ventilation is so extremely important. It also facilitates assistance to or control of respiration.

6. Maintenance of Anesthesia

Cyclopropane and oxygen are administered in a closed carbon dioxide absorption system by either the intermittent or continuous flow technique as the anesthetist prefers. The former is most often employed in our department.

A depth of anesthesia is maintained only just sufficient to produce a quiet operative field and to prevent reflex coughing during dissection about the trachea and bronchi.

Respiration is assisted or controlled to overcome the depressant effect of cyclopropane on the respiration, and to prevent collapse of the lungs, as one or both pleural cavities are not infrequently opened during the surgery.

Arrythmia has been an infrequent occurrence. More vigorous attention to ventilation will usually cause it to disappear if it reflects inadequate ventilation. If it does not the anesthesia is lightened or if this fails the cyclopropane should be discontinued altogether. This latter step has never been necessary in our cases.

Vital signs are rigorously observed, but aside from the usual mild bradycardia and hypertension common with cyclopropane, little change is usually seen.

7. Regulation of Neostigmine Infusion

To insure correct flow at the predetermined rate the intravenous infusion is frequently observed for proper drip rate. No objective signs will exist during anesthesia to verify that this rate is correct. This verification will have been made during the two hours that the infusion runs prior to anesthesia. Only if the pulse rate slows markedly under the combined influence of cyclopropane and neostigmine is consideration given to decreasing the infusion rate. Even then the usual measure is to give intravenous atropine instead.

Usually no other fluids are given except that nearly all patients receive a 500 c.c. transfusion of blood. Bleeding is sometimes marked from the sternum and an occasional patient may require greater replacement.

8. Postoperative Analgesia

Five to ten minutes before the anticipated conclusion of surgery a small dose of morphine is given. This is usually in the range of 4 to 8 mg. It is administered in two parts, one-half intravenously, the other subcutaneously.

The analgesia thus obtained is extremely useful in decreasing the severity of incisional pain as the patient recovers from anesthesia. Since little postoperative analgesia occurs with cyclopropane, the pain quickly becomes noticeable to the patient on awakening. Considerable splinting of the chest follows with resultant interference with ventilation.

9. Aspiration of Secretions

Before the endotracheal tube is removed at the conclusion of the anesthesia careful attention is paid to a thorough aspiration of the bronchi, trachea and upper airway.

10. Extubation

The anesthetic is discontinued long enough in advance of the termination of surgery so that the patient regains adequate spontaneous respiration. Most patients will awaken from anesthesia at about this time also, unless the surgery has been prolonged. Before the endotracheal tube is removed, vigorous spontaneous respirations should be present and the patient should be given a trial of three to five minutes on atmospheric air to insure that no anesthetic depression persists.

11. Verification of Respiratory Function

Before the patient is removed from the operating room he should be carefully observed for a period with the endotracheal tube removed to be absolutely certain that hypoventilation does not exist.

If it can be assumed that all respiratory depressant effect of cyclopropane is gone, and hypoventilation does occur, it can be surmised that underdosage with neostigmine has taken place. This occasionally happens despite adherence to the calculated neostigmine dosage schedule. Perhaps some patients' requirements are increased by the stress of surgery and anesthesia.

Intravenous injection of 0.5 mg. of neostigmine will usually cause the respirations to return to their normal character. Repetition of this additional neostigmine injection should be avoided or done very judiciously, as neostigmine overdosage is a serious problem. One of the effects of such overdosage is weakness of the respiratory muscles; thus, it may be difficult to detect in such a situation.

Should a patient be met who has persistence of hypoventilation after the above measures have been used, the safest approach would seem to be to continue manual or mechanical assistance to respiration until this complication subsides. Such prolonged hypoventilation has not occurred in our series following cyclopropane. It did occur, and frequently, in the first few patients we operated upon using ether.

12. Recovery Period

The patient must recover consciousness before leaving the operating room so that the anesthetist can

be certain that he will have all his protective reflexes.

The average patient has recovered to such a condition that resumption of the oral route of neostigmine administration is possible before leaving the recovery room.

All measures which are usually taken during recovery from anesthesia to protect the patient against the hazards of vomiting and aspiration, hypoventilation, obstruction of the airway, and the development of surgical shock apply with even greater force to the myasthenic individual.

Postoperative Care

It cannot be overemphasized that respiratory problems are the major hurdle over which post-thymectomy patients must be taken. Because of their muscular weakness, the lack of respiratory reserve is amazing and, at times, frightening.

No effort should be spared to be prepared for all possible respiratory complications. It is only through the development of a routine in this preparation for the postoperative period, and a thorough understanding of all the possible emergencies which may arise that these patients may be successfully carried through this most critical time. All concerned in their care; surgeon, neurologist, anesthetist, floor and private nurses must be able to instantly recognize such problems as they develop. Further, they must have immediately at hand the means to treat these emergencies. They must have no hesitation in doing so, even in a somewhat more radical manner than might be indicated for other types of patients, bearing in mind always the slim margin which may exist between success and failure. Impaired ventilation may develop very quickly in patients with diminished respiratory reserve.

Considering the overwhelming importance of maintaining adequate ventilation we have felt it prudent to insist upon the following items of resuscitative equipment being instantly available to every postoperative myasthenic patient:

A. At the bedside:
   1. Suction
   2. Tracheotomy set
   3. Laryngoscope
   4. Endotracheal tube

B. In the close vicinity of the patient's room:
   1. Anesthesia machine, or other means of providing positive pressure respiratory assistance
   2. Oxygen

C. In the hospital, and kept free for the patient's instant needs:
   1. Bronchoscope
   2. Mechanical respirator

Paramount in importance among the respiratory difficulties are profuse and viscous secretions due to the effects of neostigmine. Often this problem is inadequately controlled by atropine, or some patients may not tolerate sufficient atropine because of its other effects. In such a situation reliance must be placed on mechanical removal of secretions for maintenance of a free airway. Frequently, the patient can be taught to keep his own airway clear with a suction catheter. If, for lack of strength, or for other reasons, he is unable to do so, this should become a constant concern of the nursing personnel. If secretions accumulate below the larynx and he is unable to clear them by coughing, tracheobronchial aspiration with a suction catheter introduced through the nose into and

below the larynx must be done. This maneuver usually requires the attendance of a physician. At the same time encouragement and manual assistance to coughing should be carried out.

If all these techniques fail, if secretions continue to accumulate and respiratory exchange appears compromised, and especially if atelectasis occurs, bronchoscopy should be undertaken without hesitation. It is our strong belief that any patient requiring bronchoscopy for removal of secretions ought then to have a tracheostomy, since the situation will likely recur. Repeated bronchoscopy is an immediate hazard due to periods of suboxygenation occurring while it is being done and because of the physical exhaustion which it produces. It is also a long range hazard, since repeated bronchoscopy causes increasing degrees of mechanical trauma to the respiratory mucosa. When the vitality of this mucosa is impaired by trauma, its cilia will fail to assist in the removal of secretions. In addition trauma may cause edema of the mucosa with narrowing of the airway.

The advantages of tracheostomy in providing an easy route for removal of secretions by suction must be balanced against the disadvantage of decreased efficiency of the coughing mechanism. The production of a strong cough is dependent upon the ability to close the glottis and perform a preliminary Valsalva maneuver which increases the intrathoracic pressure and thus the speed of the airstream when the pressure is released and the cough occurs. Tracheostomy, by denying the patient this maneuver, may actually lead to increased difficulty in raising secretions from the smaller bronchial radicles where they are beyond the reach of the suction catheter. These two factors must be borne in mind when arriving at a decision as to whether a tracheostomy will be of long range benefit. In general, the advantages of assured access to the airway seem to weigh most heavily.

An occasional patient's life may be jeopardized by extreme weakness or paralysis of the respiratory muscles, with consequent inability to carry on adequate ventilation unassisted. This may arise because of difficulties with postoperative regulation of neostigmine requirements, from exacerbation of the disease, or more rarely because of neostigmine overdosage when an unrecognized remission occurs.

Whatever the cause, mechanical assistance to respiration is lifesaving in such circumstances, and some device for providing intermittent positive pressure ventilation must be at hand. This may take the form of a simple face mask and rebreathing bag, a bellows resuscitator, an anesthetic machine, or one of the many types of mechanical ventilators now available. The type of equipment is unimportant; the ability of the available personnel to operate it efficiently is of the greatest importance. The patient must be afforded instant and effective increase in his oxygen supply. In a dire emergency, mouth-to-mouth or mouth-to-tracheostomy inflation is the most simple method, and one just as effective as the more complicated devices which can be set up and adjusted when more leisure permits. If the period of artificial assistance to respiration seems likely to be prolonged, a "tank" respirator or

other mechanical ventilator which can be attached to an endotracheal or tracheostomy tube is the most practical solution.

Conclusion

In recent years many anesthetists have come to believe that less anesthetic depression of vital processes is incurred when several drugs are employed, each in small amounts, and frequently by more than one route of administration.

This contrasts with the older approach in which anesthesia was induced and maintained with one agent, or at most two or three widely used and well proven agents.

Our experience with anesthesia in patients suffering from myasthenia gravis has resulted in a synthesis of these two lines of thought. We have succeeded best with a simple anesthetic technique. At the same time we recognize the imperative necessity for correction of specific physio-pharmacologic deficits with compounds whose actions approximate those which are lacking.

Over all our thinking has hovered the impression that in dealing with a disease which is incompletely understood, there remain many virtues in simplicity. Excessive complexity often breeds disaster.

Patients with myasthenia gravis frequently have little physiologic reserve. They will not tolerate errors in management. Only by a thorough understanding of their very special problems can a routine be developed which will permit successful surgery to be performed. The establishment of such a routine is mandatory. Variance from this regimen invites catastrophe.

The principles which we believe to be vital are:

1. Exhaustive preoperative investigation
2. Careful regulation of parenteral neostigmine dosage
3. Little or no preoperative sedation
4. Adequate atropinization
5. Cyclopropane anesthesia
6. Endotracheal intubation and respiratory assistance
7. Rapid surgery
8. Avoidance of postoperative hypoventilation
9. Vigorous removal of secretions
10. Assurance of a patent airway
11. Vigilance in diagnosis and treatment of exacerbation or remission of the disease in the postoperative period.

Summary

Myasthenia gravis has been discussed as a clinical entity, considered from the standpoint of physiology, both normal and abnormal, and the pharmacologic implications of our present state of knowledge concerning this disease have been reviewed.

A large number of operations performed upon patients with myasthenia gravis has led to the development of a certain routine of management, both anesthetic and surgical. This is set forth, together with information this technique has provided bearing upon the many hazards such patients face during surgical intervention.

Esophageal Resection in Relation to Anesthesia

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Endoscopic examination and treatment of esophageal diseases have been accomplished satisfactorily for many years. The direct surgical approach has become common in the past twenty years. This has become possible because of advances in our knowledge — one of the important advances being endotracheal anesthesia.

The esophagus occupies a space in the posterior mediastinum between the spine and the heart. This position emphasizes the surgical inaccessibility of the organ.

The esophagus is merely a conveyance tube from the pharynx to the stomach consisting of mucosa, two layers of muscle and minimal nerve mechanisms. It has no digestive function and when intact has few, if any, responses to stimuli that are painful in many other locations. The nerve supply is poor and patient localization of esophageal disease is poorly defined until surrounding structures become involved. The location of foreign bodies lodged in the esophagus cannot be accurately described. The patient usually points to the sternum and designates a large area as the seat of his trouble. The esophagus permits the passage of food, liquids and saliva from the mouth to the stomach.

The normal individual produces from 500 to 2000 c.c.'s of saliva in 24 hours. This liquid he swallows awake or asleep without conscious effort or knowledge of the act. If he is unable to swallow saliva, it may trickle into the tracheobronchial tree and result in pulmonary complications.

Esophageal obstruction in our institution is most commonly due to carcinoma. In a study of some 30,000 tumors at Hines V.A. Hospital over a 25 year period, esophageal carcinoma constituted 1½ to 3½ per cent of the malignancies. With minor yearly variations, the ratio has remained fairly constant. It is of interest that carcinoma of the lung has steadily risen from 1%, 25 years ago, to 22%, 5 years ago. This fact is not pertinent to the subject at hand but demands sober contemplation.

We[1] analyzed 100 consecutive autopsied cases of carcinoma of the esophagus who died prior to the era of esophageal resection. 72 of these died as a direct result of pulmonary complications. These were bronchopneumonia, lung abscess, bronchiectasis, empyema, tuberculosis, and

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were the result of food, liquid, or saliva entering the tracheobronchial tree during unsuccessful swallowing.

The realization that a foreign body or tumor stimulates the esophagosalivary reflex to produce more saliva and large quantities of mucoid secretion by the esophagus itself is being more generally accepted.[2, 3] We have found that a nasogastric tube stimulates the reflex with an increase of salivation and esophageal secretion. The volume of liquid stimulated in this way averages 750 c.c./24 hours.

In general, anesthesia in esophageal surgery resembles any other intrathoracic procedure with a few added hazards.

If the patient is operated from the left sided approach in the standard thoracotomy position, he is lying on his right side and breathing with his right side so that he must lift the weight of his body with each breath (Figure 1). The late Evarts Graham used to comment that the standard thoracotomy position is probably the most unphysiologic of all positions. Nevertheless this position must be used in certain situations and is preferred by some surgeons for most esophageal work. The oxygenation of the patient is not easy and an alert capable anesthetist must be in charge.

If the patient is operated from the right side (it is usual to have the patient placed on his back but tilted toward the left 30° to 45°) (Figure 2), his physiology is less disturbed and he is more easily controlled by the anesthetist.

If a right sided approach is used

Figure 1. Classical left thoracotomy incision with patient lying on his right side.

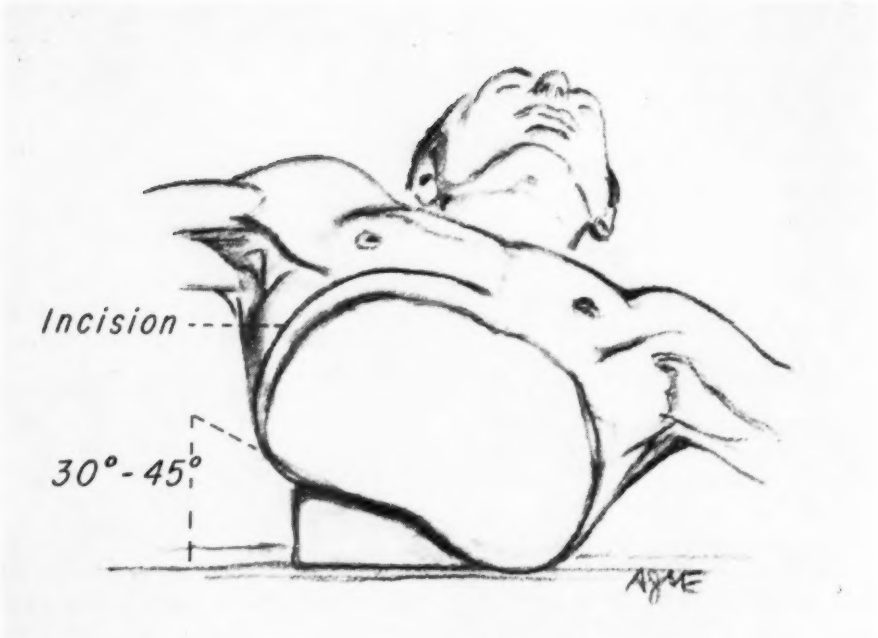

Figure 2. Right sided approach to some esophageal lesions with the patient tilted as shown.

a separate abdominal incision must be used to mobilize stomach or colon for esophageal replacement. It becomes apparent that esophageal surgery is major and potentially shocking regardless of the position used.

I have believed but I do not have actual proof that the changing of position following surgery is a shocking procedure. In the past in abdomino-perineal resections—we performed the abdominal surgery, closed the abdomen, and then turned the patient on his face to do the perineal work. Shock was frequently present and I believe the trauma of turning the patient cost us two units of blood. In recent years, we have been placing the patient in position for the abdominal work and the perineal work so that adjustments are not necessary. My impression is that the patients are better and shock is reduced.

It has seemed to me that changing the position of the patient from the left thoracotomy approach to the supine position following surgery assesses us one unit of blood. The right sided approach has not impressed me as being as costly. I must again stress that although I believe the right sided approach is less shocking, the left sided approach is easier for the surgeon and in many cases the preferable method in order to attack the disease in a particular case.

Another phase of esophageal resection cases is that their nutrition is poor. These unfortunate individuals have eliminated one food after another—first solids, later thick liquids, and finally are attempting to live on

clear liquids. The albumin/globulin ratio is often reversed, the vitamins are depleted, and they are slowly starving to death. They therefore tolerate major surgery less well than other emaciated individuals.

These people are exhausted because of lack of sleep. Because of the accumulation of saliva and esophageal secretions in the throat and inhalation of these fluids into the lung, they may sleep for one or two hours and then wake with a paroxysm of coughing. This coughing episode lasts about 20 minutes and then they fall asleep again—to repeat the coughing spell every one to two hours. Their days are fairly comfortable but their nights are miserable. I hope I am painting a horror picture because that is the way these patients exist.

What do I expect from the anesthetist? These things are basic in all anesthesia but more difficult to correct than with most other conditions.

A clear airway is essential with frequent aspiration of the pharynx as the accumulation of fluid is more rapid than usually expected (Figure 3). Fluids to be given with blood, vitamins, and glucose as the main ingredients (Figure 4). I mention amino acids merely to condemn. Emulsified fats are coming into vogue now but only time will tell whether they are of value.

I believe that proper nutrition is obtained by eating foods that look attractive with an appetizing taste. Enzyme secretions for digestion depend on psychic effects as well as chemical ones and we cannot substi-

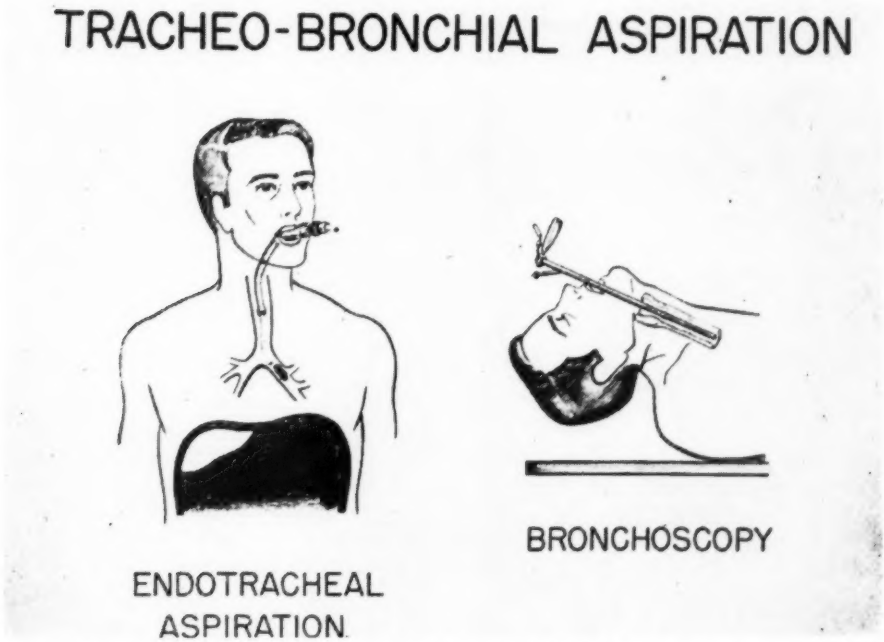

Figure 3. Tracheobronchial toilet to be accomplished throughout any surgical procedure and postoperatively.

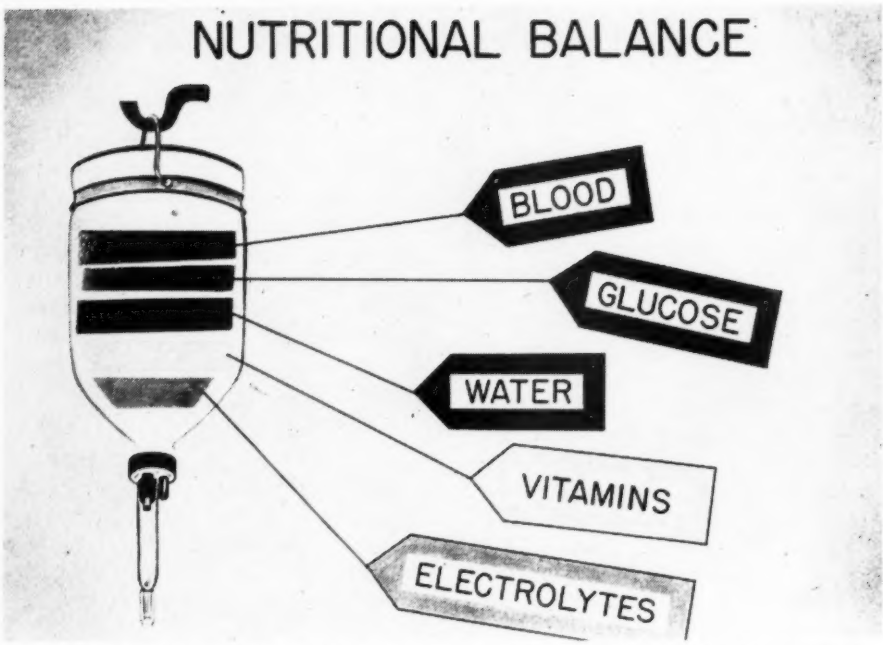

Figure 4. Nutritional balance to be improved with particular emphasis on proteins (blood) and vitamin C.

tute the intravenous bottle for a well relished steak.

In esophageal surgery we have a real problem because nutrition is so hard to maintain. It may be advisable to do a relatively minor procedure such as producing a salivary fistula in the neck—we call it a spit fistula —and a simple gastrostomy and then start feeding and X-ray therapy. Later the possibility of surgical attack can be reassessed.

Feeding of milk and protein mixtures by gastrostomy tube must be carefully done. If feeding is too rapid, diarrhea frequently occurs. 40 drops per minute is a fair rate and will usually be tolerated comfortably.

I think there are two periods when things occur that I resent and probably can be helped by the anesthetist. The first is after the patient is asleep and intubated and is to be positioned. If the attendants and house staff are careless, they j o s t l e the patient roughly and promote shock. This is particularly true if help is scarce. Smooth firm changing of positions is the result we should attempt to attain.

The other period I worry about is when the patient is transferred from the table to the cart or preferably his bed. Everyone is tired and the patient is often tossed to his bed like a sack of wheat. Our neuro-surgical colleagues treat a patient very gently because their hemostasis and work is more delicate and more easily disrupted or destroyed. Esophageal work will probably not be ruined by rough

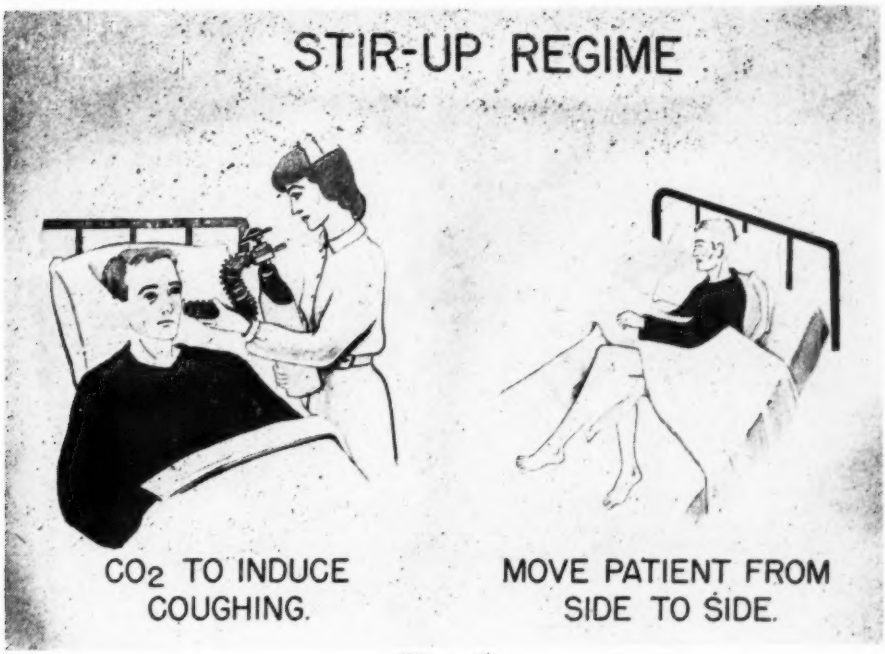

Figure 5

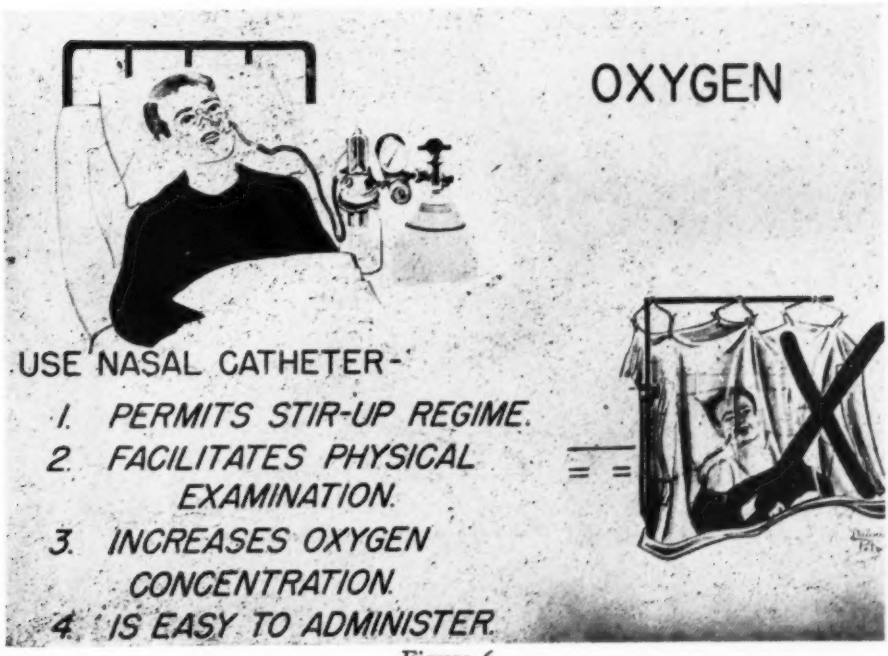

Figure 6

handling but shock will be increased by this inhuman treatment.

A postoperative period is an important one and I wish to stress a few points.

The "stir-up" regime (Figure 5) cleans out the tracheobronchial tree and prevents pulmonary complications as well as rendering thromboembolic accidents less likely.

Oxygen is to be used with caution (Figure 6) and if used is to be given by catheter. Oxygen tents are to be avoided as they interfere with patient care. We feel that attending personnel tend to neglect the patient in a tent, because they do not wish to disturb the carefully arranged drapery. They leave the patient in the care of a machine instead of retaining patient care themselves.

Sedation (Figure 7) is to be given sparingly. Intercostal blocks permit the patient to continue coughing and deep breathing with decreased pain. Finally antibiotics are to be used but will not supplant careful technique and sensible humane postoperative attention by all concerned (Figure 8).

What I have said is trite and fairly bromidic, but needs emphasis regularly.

Summary

In summary, I have pointed out that the patient with esophageal disease differs from others in the following ways:

1. He cannot swallow his saliva.
2. He aspirates saliva and develops pulmonary complications frequently.

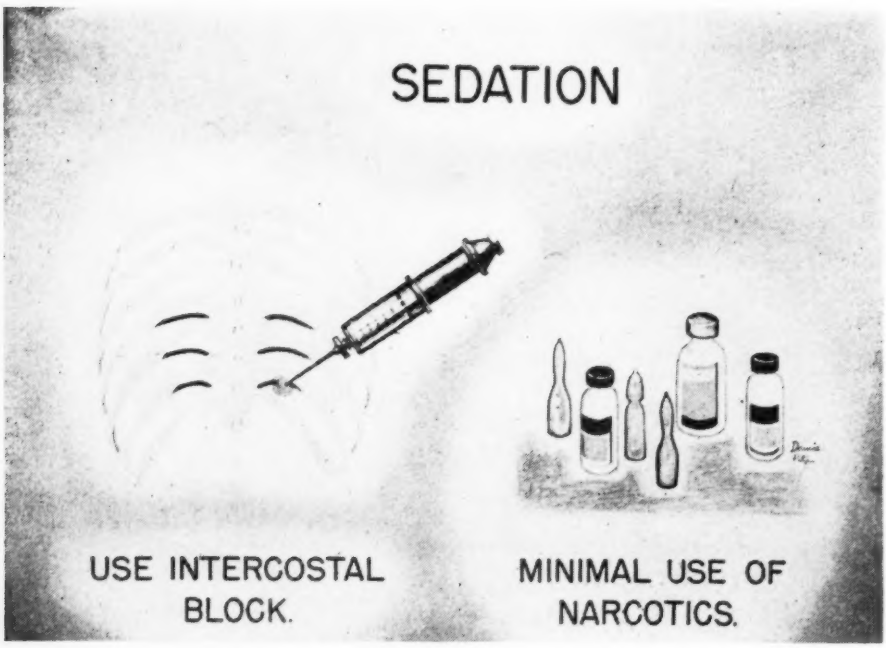

Figure 7

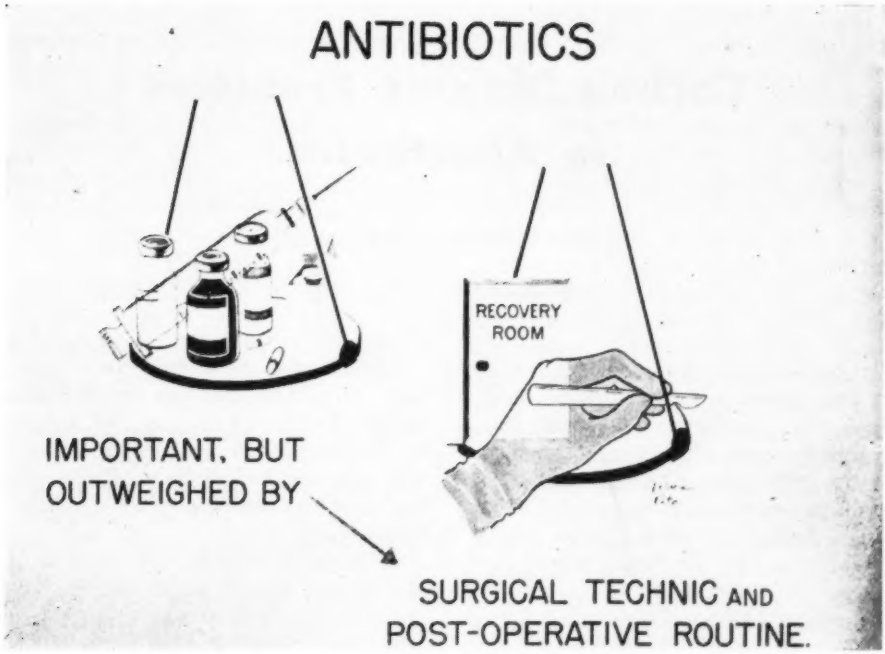

Figure 8

3. He tolerates movement under anesthesia poorly.
4. He is deficient nutritionally—proteins, vitamins — being his most important deficits.
5. He must be handled carefully before and after anesthesia.

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Carbon Dioxide Problems in Anesthesia

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Improvement in anesthesia service in this era of giant strides in medicine must, perforce, depend at least partially upon communication between those working in the field. This communication is fostered in no small degree by such active organizations as the Tri-State Nurse Anesthetists Assembly.

I would like to discuss what I feel is becoming a major problem in the field of anesthesia — the control of carbon dioxide homeostasis. In the good old days not too much attention was given to such problems—it sufficed alone that the patient was breathing during the procedure. In those days, however, procedures were short, surgeons were swift, and the patient, of a necessity, more stalwart. Today—and tomorrow—it is a different matter. The amount of attention being given to this problem is self evident from the most perfunctory perusal of the literature today. Attention is being given on the basic levels to a greater understanding of the control and problems of carbon dioxide, and on a more practical level to the machines and methods of doing something about it.

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Presented at the annual meeting, Tri-State Assembly of Nurse Anesthetists, Chicago, April 28, 1958.

One of the most important functions of respiration is the maintenance, within rather narrow limits, of the proper concentrations of oxygen and carbon dioxide within the body. This briefly is known as homeostasis.

Respiration is controlled by one of the many homeostatic mechanisms with which all animals are supplied. The usual plan for such a system is one of sensing elements, an afferent nervous pathway, a central governor, an efferent nervous pathway and an effector mechanism. The important sensing mechanisms are found in the walls of the arteries, in particular the carotid and aortic bodies. These are responsive to minute variations in the partial pressure of carbon dioxide and oxygen, and to changes in the pH of the blood. Changes in these elements are transmitted to the governing centers in the brain stem over the afferent nervous pathways. These centers act on this information to vary the synchrony, rapidity, and duration of the stream of impulses which is being constantly sent out over the efferent pathways to the affector mechanisms, the muscles of respiration. Here, at the neuromuscular junction, a greater or lesser amount of acetylcholine is released, according to the variations in the stream of nervous impulses. This in

turn affects the strength of the resulting contraction of the respiratory muscles.

Thus, the minute volume of respiration is adjusted to oppose any tendency of the content of oxygen, carbon dioxide or the pH to wander from fairly rigidly fixed normal values.

Of the three elements contributing to the control of respiration, the most important is carbon dioxide. In most cases, a level of ventilation sufficient to maintain carbon dioxide homeostasis is more than adequate to maintain oxygenation of the tissues. Only in the laboratory and in certain disease processes does this fail to be true. Secondly, the pH is inextricably tied to the partial pressure of carbon dioxide through the bicarbonate ion buffering system of the blood, by far the most important buffer system of the body. Thirdly, carbon dioxide seems to be the primary stimulant to respiratory effort, oxygen exerting an influence via the aortic body only in the presence of hypoxia.

Aside from its effects on respiration, failure of carbon dioxide homeostasis has other important effects on the general body physiology. Moderate concentration—up to 6% in the inspired atmosphere — causes an increase in cardiac output, generally thru an effect on the vasomotor center with an increased pulse rate and blood pressure. However, a further increase in concentration will cause a depression of these centers with a decline in blood pressure and a fall in cardiac output. In addition, both directly and via stimulation of the vagus center, there is an increased irritability of the cardiac musculature, resulting in cardiac arrythmias and cardiac failure. There is a depressing effect on the cerebral cortex. In dogs a 30% concentration will produce anesthesia in one minute. Convulsions frequently accompany this. Intracranial pressure is markedly elevated by inspired concentrations over 5%. Increasing concentrations favor dissociation of oxyhemoglobin, a very essential and good thing when limited to the peripheral tissues, but detrimental when occurring on the pulmonary side of the circulation, particularly when using low oxygen techniques of anesthesia such as nitrous oxide-oxygen.

These figures in some cases may seem like relatively high concentrations. Normally the amount of carbon dioxide in the atmosphere amounts to 0.03%. However, this figure in approximately 4½% of the expired air, and with rebreathing techniques in anesthesia may very rapidly mount to dangerous levels. It should be firmly fixed in mind at this point that the methods of carbon dioxide absorption in use in anesthesia today are generally inefficient. This inefficiency increases in direct proportion to the time of use, and in inverse proportion to the care with which the absorbing device is used.

This has been a very brief resume of the physiology of carbon dioxide. The control of carbon dioxide concentration within the body of a healthy man, breathing air, is a complicated business, as yet imperfectly explained. The control of carbon dioxide within the body of an anesthetized patient must in many ways be more difficult to elucidate.

There are a multitude of factors introduced to complicate this picture with the induction of anesthesia. The gaseous environment is no longer the

constant atmosphere but is a variable mixture of gases delivered by a machine which has perplexing characteristics of resistance to airflow and provides an addition to the patient's own dead space. Furthermore, the body physiology is altered by the drugs administered. The surgical procedure, the position of the patient on the table, and the patient's disease all add influences which may alter and confuse the mechanisms of carbon dioxide homeostasis.

Obviously, the position of the patient on the table has a profound influence on the ventilatory efficiency of the patient, and thus upon the accumulation of carbon dioxide. The most satisfactory position for ventilation is upright—few surgical procedures are performed in this position. There is a slight but definite decrease in minute respiratory volume on the assumption of the supine position, due no doubt to the pressure of the abdominal contents on the diaphragm as well as a slight effect of gravity on the normal lifting action of the chest wall. This effect of the abdominal contents is much magnified by the assumption of the Trendelenburg position and even to some degree by placing the legs in lithotomy. The use of the kidney and gall bladder rests exaggerates this deleterious effect, while turning the patient to the prone position decreases the minute respiratory volume by as much as 60%. All these variants have the sum effect of increasing the accumulation of carbon dioxide in the body.

The influence of the surgical procedure is easily appreciated when the chest is opened for thoracic and cardiac procedures. However, abdominal surgery with its incessant demand for muscular relaxation also results in increased carbon dioxide retention.

The patient with pulmonary disease of any type will have an increased tendency to carbon dioxide accumulation as well as toward hypoxia, as will the patient with arthritis of the spine resulting in a fixed rib cage. The diabetic with a tendency toward acidosis will complicate the picture of carbon dioxide accumulation. Even under spinal anesthesia there is frequently a reduction in minute respiratory volume and an increase in body carbon dioxide levels.

To digress for a moment there is one point where increasing the efficiency of ventilation will have an opposite effect than that which we desire. This is in the patient with chronic pulmonary emphysema who has become acclimated to a low oxygen saturation and a higher than normal carbon dioxide content. This patient's chief respiratory stimulant is hypoxia, not carbon dioxide excess as in the normal individual. Here, increasing the oxygen saturation of the blood will cause a diminution of respiratory effort. These patients generally should not receive oxygen postoperatively unless other signs of increasing hypoxia should intervene.

Even our equipment for the administration of anesthesia plays its part in increasing carbon dioxide levels, particularly in children. Any anesthetic mask, even that for open drop ether, increases the effective dead space of the patient, placing a greater demand on ventilation to maintain the same levels of carbon dioxide as before. Particularly is this true in the use of adult equipment on children and the use of improper sized soda lime canisters in to and

fro techniques. The resistance to respiration from even small degrees of airway obstruction adds its bit to the ventilatory load of the patient.

Even the agents themselves add to the picture. It has been shown that the premedicant dose of atropine may cause an increase in the anatomical dead space.

All anesthetic agents are known to depress the response of the respiratory center to carbon dioxide. Ether is unique among anesthetics in that it alone, in light or moderate degrees of anesthesia, may increase minute respiratory volume, despite a very real respiratory center depression. And, in deeper levels of anesthesia even this agent produces profound general depression of carbon dioxide homeostasis. In the awake patient, over ventilation occurs in response to increased carbon dioxide concentrations minimizing any change in the alveolar air. However, when general anesthesia intervenes to reduce the liveliness of this response, accumulation of carbon dioxide readily occurs.

The use of muscle relaxants, hailed as perhaps the outstanding advance in anesthesia in the past fifty years, has introduced a new problem. Not only is the patient's central mechanism depressed by the anesthetic agent, but the effector mechanism is now impaired by neuromuscular blockade, thus compounding the failure of homeostatic mechanisms. Beecher and Todd[2] have obliquely approached this problem in their controversial study of deaths associated with anesthesia in 1956. They found that one out of every 420 patients receiving a muscle relaxant died. However, other figures in their study suggest the real issues behind this startling figure. In the study 14,000 patients out of the total receiving general anesthetics were noted to have received assisted or controlled respirations. However, 44,000 patients received relaxants. This leaves a total of some 30,000 patients receiving relaxants and not, presumably, any assistance to respiration.

There may be some historical explanation for this failure to perform an act that is generally recognized as essential. In the early days of muscle relaxants, they were often used in small or moderate doses and usually some respiratory movement persisted. Because it did, the anesthetist refrained from giving artificial respiration. However, in the past 15 years, the complexity of new surgical techniques has increased the duration of many procedures and extended the benefits of surgery to a large group of poor risk patients who do not have the reserve to encompass a failure of carbon dioxide homeostasis. Along with this, the duties of the anesthetist have become increasingly complex. Blood and a complexity of various intravenous preparations must be administered. Voluminous records must be kept. The anesthetist is even increasingly looked upon to adjust the operating room lights, and act as general major domo in the theater. Thus, a bad habit in the beginning has been made worse by a lack of time and attention.

What is the answer to this problem? Recently, on a trip to Mexico, I was delighted to find the anesthetist had a full time assistant at every operation. This is being seen more frequently here especially during the more complex surgical procedures. Another suggestion receiving more

and more favor is the use of mechanical ventilators. There are drawbacks to these devices, admittedly, but each year improvements in the design and mechanical efficiency of these ventilators are seen. In the final analysis they are frequently an improvement over the anesthetist's hand, if for no other reason than the fact that they do not get tired on a long case nor do they have to stop to attend to a blood transfusion or take a blood pressure.

At any rate an increasing attention must be given to adequate artificial respiration during anesthesia. This thought, however, bears some further exploration. Work done in the past few years has suggested that artificial respiration may not be as efficient as an equivalent amount of spontaneous respiration. It has been found in some cases that although an apparently adequate amount of air was passed into the lungs each minute artificially, the oxygen saturation of the blood remained low. This can be corrected by increasing the amount of oxygen in the inspired atmosphere, but the effects on carbon dioxide accumulation are another problem. This, it has been suggested, relates to the uneven ventilation of the lungs by artificial methods. A deleterious effect on pulmonary circulation has been suggested as well during artificial respiration. Much more study is needed on this particular subject. Beecher,[1] once again, has suggested that a given minute volume of artificial respiration would not remove carbon dioxide as effectively as the same volume of spontaneous respiration. Lucas and Milne[11] found that respiratory acidosis developed in some patients despite minute respiratory volumes of 10 liters per minute. Dobkin[6] has concluded that artificial respiration can adequately eliminate carbon dioxide if minute volumes are increased.

Certainly during surgical pneumothorax, increased minute volumes are vitally necessary. This is not so serious, though, because artificial respiration here is indispensible. However, during abdominal surgery this can be eliminated and thus the patient is deprived of many advantages and placed in jeopardy.

One word of caution, however, is indicated. It is often assumed that over-ventilation is much less dangerous than under-ventilation. In general this is true, if for no other reason than that the former supplies an overabundance of oxygen. However, recent work has suggested that respiratory alkalosis may lead to central nervous system damage by initiating intracranial vasoconstriction and local tissue hypoxia. It has certainly been a common experience of anesthetists that vigorous ventilation while using nitrous oxide-oxygen techniques has seemed to produce a deeper level of anesthesia. However, manual compression of the rebreathing bag is unlikely to produce ventilation in excess of 14 liters per minute. Undoubtedly this may have some effect on carbon dioxide levels but the effect is limited. This, however, may become a more serious problem with the use of mechanical ventilators.

The answer to this problem is not easy. In theory, it is simple. We need some method of constantly monitoring the carbon dioxide levels in the patient and, in turn, of assisting or controlling his ventilation so that the levels remain constant, despite the loss of the individual's normal physiological control of homeostasis.

Estimation of oxygen saturation by the patient's appearance is subjective and unreliable when the hemoglobin concentration varies from normal levels. Oximetry is tricky and often so much time is consumed in attending the oximeter that the patient is neglected. Furthermore, as we have pointed out earlier, oxygenation may be perfectly satisfactory despite gross changes in carbon dioxide levels.

Therefore, the estimation of carbon dioxide levels is more interesting and important. Direct estimation by tonometry is difficult for clinical application. It can be determined relatively easily by the study of plasma carbon dioxide concentration and pH, but this requires a competent and complex laboratory setup and further, does not give us information quick enough to allow for minute to minute control of variations. Similarly, the determination of alveolar concentration by gas analysis in the laboratory is slow. Currently two physical methods of carbon dioxide analysis are available: infra red and sonic gas analysis. Both these are quick and accurate methods and can provide a constant reading estimation of carbon dioxide content. However, they are expensive and somewhat tricky to operate. The search for a simple, accurate, and rapid method of determining carbon dioxide levels is being carried on today in a number of institutions and it is felt that such a machine will soon be available. However, availability and use are two different things.

In the final analysis, our bodies are finely tuned mechanisms for maintaining the status quo. One of the more important of the homeostatic mechanisms is that for maintaining the carbon dioxide content of the body within very narrow limits. We in anesthesia are confronted at every turn by factors which upset this mechanism—factors which include the patient and his diseases, the surgeon and the positions he requires and even more important, by the anesthetist and his drugs and equipment and methods. There is no clear cut answer to the problem of carbon dioxide accumulation in anesthesia. We can only say today that the answer lies in increasing awareness of this problem and close attention to the ventilatory needs of the patient. This must of necessity mean an increased awareness of the absolute necessity of assisting or controlling respirations in the anesthetized patient, particularly when muscle relaxants are used. Just beyond the horizon is the possibility of the development of machines for the simple and efficient monitoring of carbon dioxide levels, and the providing of efficient ventilation and the efficient absorption of carbon dioxide.

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(Continued on page 295)

Anesthesia Problems in Pediatric Surgery

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More than in many of the surgical fields, the details of anesthetic management must be of constant concern in pediatric surgery. In the older group of children some of the easiest and safest anesthetics may be given but in the very young, in whom a large percentage of our work is done, no anesthetic can be considered really easy or safe. In these young patients the surgeon must be constantly aware of the way things are going at the head of the table and must try to set the pace of the operation to make the anesthetist's job as easy as possible.

I have included this table, not to point with pride to those patients that we are able to save, but to emphasize the fact that we lose so many of them. To improve these statistics will take all the skill that can be mustered by every member of the team. We must try to understand what makes the increased hazards before we can overcome them.

What makes the child different from the adult undergoing anesthesia?

First, size is the obvious difference. To the anesthetist this means a small face to which it is difficult to adjust a close fitting face mask. It means a small trachea which may become easily occluded by a very small amount of mucus or vomitus and which is difficult to intubate without considerable experience. The trachea of the newborn infant has a diameter of only 4-5.5 mm. and by age 2 this has grown to only 7.5-8 mm., as compared to the 16-22 mm. trachea of

Operation	Mortality Rate
1. Tracheo-esophageal fistula	30-35%
2. Intestinal stenosis	30-40%
3. Omphalocele	35-40%
4. Diaphragmatic hernia	11%
5. Premature with perforated ileum	20-25%

General Considerations

We could perhaps discuss specific cases but I think it best to look at the general problems encountered in children.

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the adult. In addition to having small air passages the respiratory physiology varies considerably. The tidal volume of the newborn and very young infant may average only 18-25 cc. and the respiratory rate is about 45 per minute. The minute volume will be 700-1000 cc. These figures are important to know when assisting or taking over respirations and when using anesthetic techniques such as those involving the Y tube. The breathing of the infant is largely diaphragmatic as opposed to the thoracic breathing of adults, so that a distended abdomen or pressure on the abdomen during surgery may greatly restrict the respiratory efficiency.

The blood volume of the infant is roughly proportional to that of the adult per unit of weight—that is 85-100 cc./kg. But a relatively small amount of bleeding will amount to a major hemorrhage in an infant. One ounce of blood from a 6 pound baby equals one-tenth of its total blood volume and corresponds to about 600 cc. in a 150 pound adult. Not only must this be considered in assuring adequate replacement of loss but in avoiding over-replacement as pulmonary edema can quickly result.

The metabolic rate of infants is high. This results in the need for larger doses of certain drugs and anesthetic agents in proportion to size. The surface area per unit of weight is larger resulting in more labile temperature control and increased fluid loss from the skin. When this fluid loss is coupled with fluid loss from the lungs by the rapid respirations a large fluid turnover results so that during long operations fluid replacement becomes very important.

Young babies may seem to tolerate hypoxia better than older people with apparent full recovery. Let this not be a temptation to tolerate hypoxic situations, however. We feel that permanent damage to higher cerebral centers may be done which may not be apparent at the time but which may result in impaired mental development later on.

Anesthetic Management

With these differences in mind let us review, step by step, the important features of anesthetic management. The preoperative preparation of the patient is of greatest importance. The nearer to normal the patient's physiology at the beginning of anesthesia the less the likelihood of trouble. The careful anesthetist will not rely on the surgeon to have checked everything ahead of time but will follow her own preoperative check list. She should be certain that there is sufficient hemoglobin by checking the blood count. Many normal appearing infants are anemic, especially those 2-3 months of age. We consider 10 gm. is the bottom limit for safety. The pulse and blood pressure should be checked before induction is begun —the patient in impending shock may go into real shock when the anesthetic is given. If the pulse of an older child is over 120 or, of an infant is over 140 make sure the surgeon knows it before beginning the anesthesia. Fever is a real and constant danger in children and the temperature should be recorded immediately prior to any anesthetic. This is particularly important when ether is to be used. In this state, recently, one hospital reported within one year's time four ether convulsions in children who had fever during the anesthetic. Two of these children died and another had permanent severe brain damage. Experiments in ani-

mals showed consistent brain damage when ether anesthesia was coupled with fever. Other anesthetics failed to show this tendency and Pentothal® induction seemed to protect against it; however, the damage appeared to be irreversible once it had occurred. Why take a chance? There is seldom anything to be lost by spending one or several hours getting the temperature down preoperatively. One of the most effective ways to reduce the child's temperature is an oxygen tent with iced oxygen. If surgery must be begun when some fever persists it may be best to avoid using ether. Protect yourselves from such tragedies by always checking the temperature before starting anesthesia.

Premedication is a controversial subject. There are at least four good reasons for preanesthetic medication. The first is the emotional problem. Eckenhoff[1] in Philadelphia found that about 17% of the emotional disturbances found in children following hospitalization could be related to inadequate premedication. Second, the drugs tend to lower the metabolic rate thereby decreasing reflex irritability and reducing tissue use of oxygen. Third, the amount of mucus secreted is diminished. And fourth, the likelihood of vagal reflexes affecting the circulation is reduced. Much work is being done on the use of the tranquilizer drugs for premedication. I have had no experience with them and still prefer Nembutal,® atropine and either Demerol® or morphine. Proper dosage is of primary importance. I prefer to follow the table of dosage recommended by Dr. Robert Smith[2] of the Boston Children's Hospital. I think every hospital should have a published table of recommended doses on each ward. This permits the doctors who handle children only occasionally to check the doses. Also, the nurse should check this table before giving the premedication. If there is a great discrepancy between the order and the table she should check with the doctor to see if the order is correct. Such a system will lead to uniformity of premedication which will help the anesthetist to know what to expect rather than having to cope with all different degrees of sedation and atropinization. It is important too, to give the drugs time to act. Allow at least 45 minutes for the hypodermic to be absorbed.

The stomach should be empty when the patient comes to the operating room. This is not always the case even when the patient has been NPO for 6-8 hours. A small amount of gastric juice may cause severe troubles in an infant's trachea. Therefore, we believe that every infant undergoing surgery and anesthesia should have a tube passed into the stomach to empty it. If the patient has been NPO we wait until he is asleep to insert the tube. If vomiting has been a part of his illness or, of course, in the unprepared emergency patient the tube should be passed before induction is begun. Small infants have a rapid fluid turnover and will dehydrate rapidly. Therefore, we like to give them clear fluids preoperatively up until 4 hours preoperatively unless it is planned to give an intravenous. These patients in particular should have their stomachs aspirated.

In older children, chewing gum and loose teeth are a menace and should routinely be checked for before starting anesthesia.

Now, we have checked the temperature, the pulse and blood pressure

and the blood count. The premedication has had time to act and the mouth and stomach are empty—we are ready to proceed. Positioning the infant is important. Remember that the breathing is abdominal. Place the baby so that there is as little restriction of abdominal movement as possible. For operations on the perineum the face down position is less desirable than the lithotomy position which may be easily set up by tying the legs up to an anesthesia screen. We must always be very careful in restraining these small patients as pressure areas develop very easily and have led to many unpleasant and unwarranted injuries.

The choice of anesthetic agent and technique I will pass over lightly. For those who do not routinely handle infants I am sure that open drop ether is the safest and most satisfactory agent. In practiced hands many other agents have distinct advantages. As to technique, the endotracheal tube is the main point in question. Some recommend endotracheal anesthesia for practically every operation in children. I have seen this work beautifully in many hundreds of children. On the other hand, in the not too distant past, there have been places where up to 10% of infants having endotracheal anesthesia have required tracheotomy postoperatively. This, of course, cannot be tolerated. To avoid complications of this technique several things are important. Skill in inserting the tube quickly and with minimum trauma is one of the main factors and comes only with practice. A knowledge of the proper size tube will save a lot of time and effort. Perhaps a chart should be available in every operating room which gives the diameter and length of tube necessary for children of various sizes. I think this chart should state the average diameter and length of the trachea in these patients. Next to be considered is the care of the tubes. Sterile technique must be used. Anesthetists have actually been observed to lubricate endotracheal tubes with their tongues. They deserve the resulting complications. The lubricant should be sterile. A series of near fatal complications following the use of a lubricating jelly contaminated with bacteria occurred in one Children's Hospital in Philadelphia only a few years ago. And, a final word about endotracheal tubes. It is considered unwise to leave a suction catheter through the tube as it is being removed. Trauma to the cords by strong suction has caused severe laryngospasm in these tiny patients.

During surgery intravenous fluids must be carefully controlled. The anesthetist is in the best position to supervise this. It is important to know how much fluid should be given to children of various sizes. A normal child will tolerate the rapid infusion of about 10 cc./lb. I think this is a good figure to keep in mind. One should not plan to exceed this volume of fluid intravenously during the ordinary operation. This figure also applies to blood transfusion. Of course, when considerable blood is lost it should be carefully measured and accurately replaced. In most small babies we use cut downs and it is possible for fluids to run in very rapidly. This can be very dangerous if the intravenous is not watched very carefully. It is a good rule to start with no more fluid than the patient can safely tolerate rapidly. That is—never put more than 10 cc./lb. in the intravenous bottle at a time.

We have mentioned the dangers of fever during anesthesia. Patients with normal temperature frequently develop very high fever during surgery. Under 6 months of age the reverse is frequently true and hypothermia may develop. This later may not be harmful unless it is extreme. In any event it should be controlled if possible. Bigler and McQuiston[3] have developed a water mattress on which children are placed during anesthesia. I would like to see this used routinely with a constant recording thermometer in place to aid in using it.

Monitoring the pulse and blood pressure of infants during anesthesia is difficult. The easiest way to follow the pulse is by a stethoscope strapped to the chest. This should be done in every case. Using the small infant cuff, the blood pressure can also be followed and I heartily recommend it.

When surgery is completed, the danger may be at its greatest. Many infants are lost from postoperative shock and especially from aspiration of vomitus. The stomach should be aspirated routinely at the end of anesthesia. The patient must then be carefully followed until fully reacted, preferably in a recovery room well equipped with laryngoscopes, endotracheal tubes, suction machines and someone who knows how to use them.

I have left until last the discussion of a very important aspect of pediatric anesthesia — the psychologic aspect. It is estimated that 20 to 50% of children have emotional disturbances following hospitalization and a number of these reactions may be directly related to anesthesia or surgery. Included in the types of reaction are regressive behavior, loss of bowel and bladder training habits, excessive fears, sleep disturbances, night terrors, tics, negativism, temper tantrums, and destructive behavior. These reactions are most common in the under 4 age group. Most of them last less than 3 months—only 15% last over 6 months. Some disturbances may go on for 3 or 4 years. however, and permanent damage may result. Much can be done by doctor and parents to better the adjustment of the child to the experience. What can the anesthetist do? First, adequate premedication can be insisted upon. Next, the anesthetist can visit the patient in his room preoperatively. This not only permits better planning of the anesthetic, but lets the child find a familiar face and voice in the strange, cold operating room to help reduce his fears. Induction by the use of space helmets, dolls with concealed tubes carrying anesthetic gases, and other methods are in use. The important thing is that the anesthetist realizes the possibilities of mental trauma and makes every effort to make the experience as pleasant as possible.

Summary

In summary then, I emphasize the need for considering the infant and child as a special problem, and not merely a small adult. We have been able here to touch very lightly on the many problems. I urge you who are giving anesthesia to children to read carefully the many articles available on the subject and to use every available safety measure on every single case to give these little ones their best chance for survival.

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Radiation Hazards in Diagnostic Radiology

Matthew W. Elson, M.D.*
Columbus, Ohio

"Stress must be laid once more upon the importance of avoiding all unnecessary exposure to x-rays. The rays have an undoubted power to produce sterility in both sexes. It is advisable to have some idea of the exposure we may give the patient during the course of an x-ray examination without causing him to suffer any ill effects." The forementioned is a quotation which sounds like an admonition of 1959 but in reality is taken from a textbook, *X-Ray Diagnosis and Treatment* published in 1912. By 1927 over 120 papers had been published on the physics of protection alone.

Our body is made up of many types of tissue and each type of tissue is composed of countless cells. What happens when an x-ray strikes a cell? There are several modes of radiation action on cells; one mode is by target or point injury. Like a bullet striking a vital spot, an x-ray may strike a sensitive system in a cell and produce cellular death. It follows that multiple minor injuries to multiple areas of the cell may not be immediately lethal but may eventually produce cell death. Similarly, an x-ray may produce ionization in the chromosome of the cell nucleus conceivably causing genetic changes that could be passed on to subsequent generations.

As a by-product of concern over the effects of fallout from thermonuclear weapon tests, or the worse eventuality of thermonuclear warfare, public attention has again been directed to the hazards of the medical use of ionizing radiation.

X-rays or any type of ionizing radiation when used to excess produces undesirable biological change. Indeed, this was widely recognized in the early decades of the century, when physicians learned that distressing and often irreversible changes developed in those men who tested the efficiency of fluoroscopic equipment each day by placing their hands in the radiant beams to observe the quality of the images produced thereby. As a result of these experiences, physicians have long since learned to take precautions against very large doses of radiation to which their predecessors often unwisely subjected themselves.

By the use of lead aprons and gloves, radiation exposure during fluoroscopy has been reduced to what appears to be insignificant levels. Also, by the use of diaphragms, cones,

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* Assistant Professor, Department of Radiology, University Hospital, Columbus, Ohio.
Presented at the annual meeting, Ohio Association of Nurse Anesthetists, Columbus, April 8, 1959.
The bibliography used for this article will be supplied on request.

filters, and fast films and intensifying screens, patient exposure has been brought under apparently safe control. It should be pointed out that the measures currently used by physicians have been principally designed to avoid the somatic effects experienced by early radiation workers.

Since the quantities of radiation against which these precautions were directed were large, these precautions may not necessarily be adequate for the protection of patients and radiation workers against damage which inherently requires only a small amount of radiant dose for its production.

It is indeed this fact, which more than anything else, I believe, requires that physicians examine very carefully their position concerning radiation safety. This is particularly so when experience has shown, during the past few years, that many physicians have often been careless in the application of accepted safe operating principles.

The somatic effects of protracted doses of ionizing radiation may be divided into two principle categories: (a) those produced by relatively large doses, and (b) those produced by medium and small doses.

In the large dose category, the principle manifestations are those leading to carcinoma of the skin. With today's techniques in diagnostic radiology this effect does not appear to be of great concern. Great care should be exercised in balancing radiation hazard against medical benefit, wherever radiation is contemplated therapeutically in the treatment of such benign lesions as skin inflammation, bursitis, and spondylitis.

The principle somatic effect of protracted doses of radiation of small or medium magnitude (1 to 100 roentgens total) is the production of such malignancies as leukemia and carcinoma of the thyroid. This relationship is evident in the study of the citizens of Nagasaki and Hiroshima who received a wide range of radiation dosage following the detonation of nuclear weapons during World War II. Also in Great Britain there is a study of the children of mothers who received diagnostic abdominal radiation during pregnancy. Here, it appears that the incidence of acute leukemia during the first ten years of life is approximately doubled in children of mothers who received such radiation. It is not infrequent in pediatric practice today for the physician to fluoroscope infants and small children whenever indications of a respiratory infection exist. Small numbers of radiographic films usually serve just as well as a fluoroscopic examination in the terms of the yield of diagnostic information; since even with a half dozen films, the radiation exposure to the individual is a small fraction of that needed in fluoroscopy, the advantages of replacing fluoroscopy with films are quite obvious. In radiography of the chest of a child, for example, seldom is the exposure more than .05 roentgens. Films of the skull or abdomen seldom take more than .5 roentgens. Hence, if it is found necessary to take ten films of the abdomen and ten films of the chest of an individual during his or her childhood, the leukogenic effect will only be a small fraction of that created by fluoroscopy.

It is the genetic effects of radiation that perhaps are of greatest impor-

tance of all. These effects are directly proportional to the radiation dosage received by the gonads. Furthermore, when a mutation occurs, it is irreversible. Also, there does not appear to be any threshold level of radiation dosage below which genetic effects do not occur. A few of the genetic changes are lethal, and the fertilized ovum will not survive gestation. Other changes may not be lethal but will be of such severity that the newborn will not be able to survive more than a few weeks or a few months. Others will be less severe, but will plague the individual with some deformity or affliction which makes life more difficult. Finally, many mutations will be recessive, and will not manifest themselves until many generations have passed and an ovum and a sperm with the same defect come together. In such a situation, one may have the range of genetic effects just cited from complete lethality to the development of an affliction which interferes with normal life processes.

The risk involved is, therefore, concerned with damage to the reproductive cells or to a developing embryo. X-ray examination of the pelvis of women who are pregnant but not aware of the fact is fraught with hazard to the embryo. There are critical periods of organ development when very low doses can cause demonstrable changes. This critical period is probably the second to the sixth week of gestation and pregnancy may be unsuspected during part of that time. The recommendation has been made that all elective radiation involving the uterus of women of childbearing age should be restricted to the two weeks following the last menstrual period, in order to preclude the possibility of fertilization having taken place. Examinations involving the lower abdomen and pelvis, while representing a small percentage of the examinations done in most offices and hospitals, account for a large percentage of the gonadal dose. What is not generally recognized is that practically every examination contributes something to the gonadal dose.

Chest microfilming or the photoroentgen survey is of particular interest because of recent criticism leveled at this type of survey procedure. The value of this has been well documented and it is important that we continue this type of examination. Although the death rate in tuberculosis has been reduced almost 100 percent in the last five years, the incidence of tuberculosis remains about the same.

Many people have jumped to the conclusion that chest microfilming may have grave genetic significance and therefore should be discontinued. As a matter of fact even in females who receive one chest microfilm a year made with the unit in common use today, the gonadal dose over a period of 30 years is very small and in males over the same 30 year period the dosage is virtually insignificant. Or visualized another way more graphically, the amount of radiation received from one chest fluorograph is equivalent to that received during a one month vacation in the Poconos Mountains.

Is there a permissible gonadal dose of radiation to which the population at large may be subjected without causing serious damage to future generations? Arbitrarily, a figure of 10 roentgens during the first 30 years of life has been chosen by knowledge-

able genetic and biological groups for this dose. This problem simplifies itself somewhat when it is recalled that the gonadal dose of an average individual during his or her reproductive life from natural sources ranges from two to five roentgens. Certainly, medical x-ray applications cannot be of great genetic importance if such examinations produce a gonadal dose which is only a small fraction of the dose from natural sources. The possibility of reducing gonadal exposure on the average to 1 roentgen or less and still retain all the important medical benefits we now enjoy seems to be good. By the careful limitation of radiation fields, the use of adequate filtration (2 mms. of aluminum or more) and by the application of screen intensification to fluoroscopy, substantial reductions in gonadal dosage can be made without curtailing medical benefits.

It behooves the radiologist and the consulting physician to work closely together to insure that the proposed x-ray examination is indicated, that the likelihood of obtaining information is greater than the risk of radiation damage, and that the patient is well prepared and the equipment is designed and used only in the safest possible manner.

Notes and Case Reports

A Syringe Holder for the Anesthetist

This article presents a design for a simply made syringe holder. It was designed to hold a syringe in a convenient position and to offer secure support.

This holder can be made with a minimum of equipment and expense. The parts needed can be obtained from most hardware stores or maintenance shops. They are listed below:

One—Aluminum bar, 1 inch by ¼ inch, 13½ inches long. (Cost: about 40 cents)

One — Spring type broom holder. (Cost: about 10 cents)

One — 6-32 screw, ¼ inch long. (Cost: about 2 cents)

One—#4 sheet metal screw, ¼ inch long. (Cost: about 2 cents)

The parts are assembled as listed below. Illustration No. 1 shows the parts before and after assembly.

1. A 1/8 inch hole is drilled through the bar 1/2 inch from the upper end.
2. A 3/16 inch hole is drilled 3/8 inch deep into the side of the bar 4-1/2 inches from the lower end.
3. Bend the bar 6 inches from the upper end to an angle of 135 degrees.
4. The spring holder is attached to the end of the bar with the 6-32 screw. The screw will tap its own thread into the 1/8 inch hole.
5. Insert the sheet metal screw into the hole in the side of the bar.

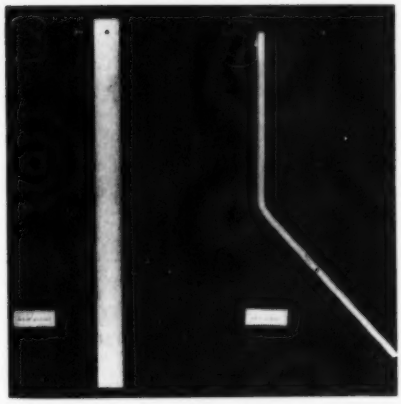

Fig. 1

This screw acts as a stop when the syringe holder is used in the sliding sockets of the operating table.

6. Spread apart the two sides of the spring holder with a pair of pliers so that a 30 cc. syringe snaps in and out easily. If the fit is too tight the syringe may be broken; if it is too loose it may not be held securely. This is an important step.

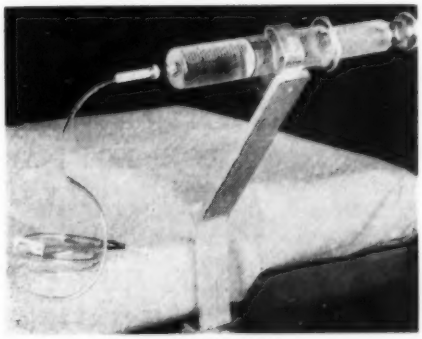

Fig. 2

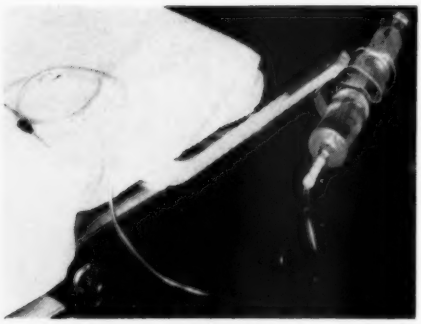

Fig. 3

Fig. 4

As illustrations No. 2, 3, and 4 demonstrate, this holder can be used in three different positions; it can be inserted into the sliding sockets along the side of the operating table, it can be placed under the mattress of the table, or under the pad of the armboard. In any of these positions it will afford secure support for the syringe, and will keep it within easy reach of the anesthetist.

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Legislation

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Malpractice Case Arising from Injection of Sodium Pentothal Should Have Been Submitted to Jury

On August 3, 1954, plaintiff, while under the care of Dr. Barr, was admitted as a patient to a hospital to determine the cause of her obesity.

In accordance with the custom at the hospital, any doctor who was free would act as anesthetist. In this case it was Dr. Marsh. According to plaintiff's testimony, Dr. Marsh first endeavored to inject the left arm and being unsuccessful there proceeded directly to inject the inner aspect of her right knee.

Dr. Barr stated that when he saw plaintiff approximately an hour after the injection she was complaining of pain in her leg and that he examined it and found a reddened area around the site of the injection which was more firm than the surrounding tissue.

Within two or three days after the injection, a thrombosis developed, and shortly thereafter at the site of the injection there appeared a "slough ulcer."

Dr. Barr testified that in the exercise of the standard of care in that community a physician would not inject sodium pentothal into a varicose vein, since the walls of such veins do not have normal tensile strength, tearing easier and not having the resistance to irritants that normal veins have. (b) Dr. Robertson, a local physician, testified that only in an emergency would he inject a varicose vein and that the standard of practice in the community forbade such an injection.

It is a matter of common knowledge among laymen that injections in the arm, as well as other portions of the body, do not ordinarily cause trouble unless unskillfully done or there is something wrong with the serum.

The conclusion that negligence is the most likely explanation of an accident or injury is not for the trial court to draw or refuse to draw so long as plaintiff has produced sufficient evidence to permit the jury to draw the inference of negligence, though the court itself would not draw that inference. The court must leave the question to the jury where reasonable men may differ as to the balance of probabilities.

A new trial was ordered for the jury to determine the question of malpractice.

(Wolfsmith v. Marsh, 9 CCH Neg. Cases 2d 508-Calif.)

Doctor Liable for Failure To Disclose Broken Catheter Left in Patient's Body

Plaintiff is an elderly Chinese woman who had been treated for a number of years by Dr. Ing as her

family physician. In March, 1954 plaintiff was taken to the hospital with a severe illness of what was afterwards diagnosed as caused by gall stones as diagnosed by Dr. Yee who was called in by Dr. Ing as a specialist; this diagnosis was also verified in a consultation with Dr. Joseph Strode.

Dr. Yee operated on March 10 for the removal of gall stones with Dr. Ing assisting. After the removal of the stones both from the gall bladder and from the duct, Dr. Yee inserted a French catheter into the wound for drainage purposes. This catheter was furnished by the hospital and examined by the doctors. After the operation both doctors Ing and Yee attended the plaintiff in the hospital and on March 25 the catheter was removed. At the time it was removed from the wound, according to Dr. Yee's testimony, he noticed that the tip of the catheter, about 1¼ inches long, which was rounded and perforated, had failed to come out with the rest of the tube.

Physicians testified at the trial that if that portion of the catheter were left in the patient's body, one of two things would happen: it would either become encysted or would follow the sinus created when it was inserted and finally extrude. The portion of the tube which remained in plaintiff's body was permitted to remain there until it finally extruded by natural process on May 1, 1955, more than a year after the operation.

While it may not be the custom, as some doctors testified, to notify a succeeding physician what has been done, the condition of the patient, and what treatment has been given, certainly Dr. Ing made no effort to notify plaintiff, her family or her succeeding doctors, so that future treatment could be intelligently followed up. In fact, he apparently made no effort after plaintiff left the hospital to keep in touch with her progress as all the doctors testified should be done. He made no calls upon plaintiff on his own initiative and at no time instructed her to return at stated intervals. There is testimony of two doctors that one reason such a wound should be watched is that an abscess might develop.

Further, taking the defendant's own testimony that plaintiff should be told at the proper time, there is sufficient evidence for the jury to decide that she should have been informed the reason the wound did not heal.

There was substantial evidence, more than a mere scintilla, for the jury to find Dr. Ing failed to give proper postoperative care and that such failure resulted in suffering and large expenditures by the plaintiff.

The jury returned a verdict in favor of defendant Dr. Yee and against defendant Dr. Ing, awarding damages against the latter in the sum of $11,000.

(Ho v. Yee et al., 9 CCH Neg. Cases 2d 992-Hawaii.)

Hospital Safety

Harriet L. Aberg, C.R.N.A.

Dental Engines

We have been asked whether or not it is safe to use the Ritter Dental Engine and foot pedal in areas where flammable anesthetics are to be used. The answer to this is best expressed in a quotation from the Ritter Company, Inc.

"If any of our Dental Engines are being used where flammable anesthetics are administered, it is without our knowledge. We have had many requests to supply our Dental Engine for such use and we have, without exception, turned down the request and explained that it is not a safe item for this use."

This is an example of the definite kind of reply from manufacturers of equipment. They are vitally concerned with the method in which their equipment is used, and are willing to advise concerning any dangers in the use of their product. Manufacturers do not want their equipment misused from any standpoint. To obtain the best and safest service, equipment should be used in the manner for which it was devised.

Annual Report
N.F.P.A. Representative

The National Fire Protection Association's Committee on Hospital Operating Rooms met January 19 and 20, 1959. Your representative attended all sessions.

The greatest accomplishment of the committee this past year is a complete rewriting of pamphlet #56 — *Recommended Safe Practice for Hospital Operating Rooms*. This was done with two main aims in mind: First, for clarification, and second, for some means of making easier the enforcement of the requirements of local authorities.

The first was brought about by rearrangement of sections into what seems to be a more logical sequence, and renumbering to correspond to the National Electrical Code.

The second was accomplished by rewording to make pamphlet #56 requirements mandatory, no longer just recommendations. The committee has also asked the N.F.P.A. Board to change the name to the mandatory title *Code for Use of Flammable Anesthetics*.

This new title is far more indicative of the content and intent of the pamphlet than the old title. Pamphlet #56 deals specifically with hazards in the use of flammable agents as anesthetics, whether administered in operating rooms, delivery rooms, out-patient departments, accident rooms, medical offices or any other place. Also, with this more accurate title, there will be better understanding why danger of infection, for instance, plays no part in ventilation requirements.

Your representative is endeavoring to answer questions concerning clothing, equipment and other subjects related to the use of flammable anesthetics in a column in the Journal devoted to this subject of Hospital Safety.

At their June meeting, the N.F.P.A. Board voted to change the title of #56 to *Code for Use of Flammable Anesthetics*. And for a few years the old title *Safe Practices for Hospital Operating Rooms* will appear in parentheses just below the new title. The price of the new pamphlet is 50c per copy and may be ordered from The National Fire Protection Association, 60 Batterymarch St., Boston 10, Mass.

Miss Aberg is A.A.N.A.'s representative on the N.F.P.A. Committee on Hospital Operating Rooms.

Any questions pertaining to hospital safety may be directed to the Executive Office. Answers will be included in this section in future issues.

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Abstracts

Okun, Ronald and Elliott, H. W.: Acute pharmacological studies of some new morphine derivatives. J. Pharmacol. & Exper. Therap. 124: 255-259 (Nov.) 1958.

"Modifications of the C-ring of the morphine molecule in the neighborhood of the alcoholic hydroxyl group have resulted in a number of potent analgetics some of which have achieved a degree of clinical acceptance. . . . Related compounds including dihydrodesoxymorphine, 6-methyldihydromorphine and 6-methyl-△-6-desoxymorphine, have been potentially useful but have either been difficult to prepare or have shown no real advantages over morphine. Rapoport and coworkers were intrigued by the structure-action relationships of these compounds and noted that all have ring C either reduced or methylated or both and the hydroxyl at C_6 is absent. They succeeded in preparing several new structural variants of this type including morphinone, 6-methylmorphine . . . 6-methyldihydrodesoxymorphine, 6-methylenedihydrodesoxymorphine and 6-methyl-7-hydroxydihydrodesoxymorphine. . . .

"Five new morphine derivatives were studied in mice with regard to analgesia, acute toxicity and gastrointestinal inhibition. 6-methylmorphine, 6-methylenedihydrodesoxymorphine, 6-methyl-7-hydroxydihydrodesoxymorphine, and 6-methyldihydrodesoxymorphine are more potent and have higher therapeutic indices than morphine. Morphinone is less potent and has a lower therapeutic index. All compounds cause less gastrointestinal motility inhibition in mice.

"6-methylenedihydrodesoxymorphine was studied in dogs with regard to analgesia, sedation, cardiovascular and gastrointestinal changes, hypothermia, miosis, and reversal of actions by nalorphine. The findings suggest it may cause analgesia in doses which have minimal side-effects."

Wilson, Michael, Perez-Arzoia, Michael and Oppenheimer, M. J.: Mephentermine and the arrhythmias. Am. J. Med. Sc. 236: 300-310 (Sept.) 1958.

"The purpose of this clinical study is to evaluate the acute response of various cardiac arrhythmias in the abnormal heart, to mephentermine sulfate. . . . Twelve patients were studied. . . . Mephentermine sulfate has an anti-arrhythmic effect upon the human diseased heart. This salutary action was especially noted in patients with ventricular ectopic systoles.

"The mechanism of action is a decrease in the refractory period, an increase in the velocity of conduction and a positive inotropic effect. In doses up to 3 mg. per kilogram administered intravenously there was no significant electrocardiographic effect in patients with chronic atrial fibrillation.

"Effective reduction in functional atrioventricular block was produced.

Supraventricular tachycardia with second degree atrioventricular block represents a contraindication to the use of mephentermine. The antifibrillatory properties of mephentermine may be of value in open heart surgery with therapeutic cardiac arrest. Theoretically, an oral preparation of mephentermine may find use as an adjuvent to digitalis in patients with chronic congestive heart failure."

Greisheimer, Esther M., Ellis, Dorothy W., Stewart, G. H., III, Makarenko, Lydia and Oppenheimer, M. J.: Cardiovascular functions under chloralose-urethane and 21-hydroxypregnane-3, 20 dione sodium succinate (Viadril). Am. J. Physiol. 190: 317-319 (Aug.) 1957.

"The first steroid anesthetic agent to be widely studied is 21-hydroxypregnane-3, 20 dione sodium succinate or Viadril. . . . One of the undesirable effects is hypotension in some patients. . . .

"Fifty-five determinations of cardiac output were made on eight mongrel dogs under chloralose-urethane. The range of cardiac index was 1.71-7.46, with a mean of 4.08 and a standard deviation of 1.45. The systolic blood pressure ranged from 141 to 231, with a mean of 202, and a standard deviation of 20. The diastolic pressure ranged from 78 to 148, with a mean of 118, and a standard deviation of 19. The mean blood pressure ranged from 100 to 173, with a mean of 146 and a standard deviation of 18.

"The peripheral resistance ranged from 2884 to 14,940, with a mean of 5709 dynes/sec/cm-^{5} and a standard deviation of 2168. The range of heart rate was 96-232, with a mean of 149 and a standard deviation of 35. The stroke index ranged from 11 to 57,

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with a mean of 27 and a standard deviation of 9. Inverted T waves were observed frequently under chloralose-urethane anesthesia.

"Forty-one determinations were made on six mongrel dogs under 21-hydroxy-pregnane-3,20 dione sodium succinate. The range of cardiac index was 1.27-11.05, with a mean of 5.15, and a standard deviation of 2.26. The systolic blood pressure ranged from 73 to 215, with a mean of 154 and a standard deviation of 39. The diastolic blood pressure ranged from 36 to 123, with a mean of 79 and a standard deviation of 28. The mean blood pressure ranged from 54 to 147, with a mean of 104 and a standard deviation of 28.

"The peripheral resistance ranged from 1517 to 8749, with a mean of 3997 dynes/sec/cm-^{5} and a standard deviation of 1916. The heart rate ranged from 135 to 210, with a mean of 172 and a standard deviation of 21. The range of stroke index was 8-59, with a mean of 29 and a standard deviation of 12. . . .

"The changes in cardiovascular functions under chloralose-urethane during successive determinations resembled those under other anesthetic agents, in that the cardiac index and stroke index increased markedly, peripheral resistance decreased markedly, but the blood pressure and heart rate showed minimal changes. The cardiovascular functions under 2,-hydroxy-pregnane-3, 20 dione sodium succinate were unsteady also. Of all the agents observed, cyclopropane seemed to be the most satisfactory agent, so far as maintaining steady cardiovascular functions in the dog is concerned.

"We found hexobarbital and 21-hydroxy-pregnane-3, 20 dione sodium succinate highly unsatisfactory as sole anesthetic agents in dogs, since the analgesic effects were minimal under the conditions."

The THIRTY-FIRST QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on May 14, 1960. The deadline for accepting completed applications including the transcripts is April 1. Notice of eligibility will be mailed about April 10.

Applications should be forwarded early enough to allow time to request transcripts and have them returned to the Executive Office before the deadline date.

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Book Reviews

Resuscitation of the Unconscious Victim. A Manual for Rescue Breathing. By Peter Safar, M.D., Chief, Department of Anesthesiology, Baltimore City Hospitals; Assistant Professor of Anesthesiology, The Johns Hopkins University School of Medicine; Clinical Associate Professor of Anesthesiology, University of Maryland School of Medicine and Martin C. McMahon, Captain, Baltimore Fire Department, Ambulance Service. Springfield, Ill., Charles C Thomas. Paper. 79 pages, illustrated, 1959. $1.75.

This concise manual presents the techniques of resuscitating unconscious persons whose airways are obstructed or who have stopped breathing.

Dr. Safar's recommendations are based on his experiences and on research at Baltimore City Hospitals.

The volume is divided into four sections dealing with asphyxia and a description of how to handle this emergency.

15 illustrations are included. A glossary and bibliography complete the work.

This practical manual will be of special interest to anesthetists. Those involved in any phase of first aid will find it a welcome addition to their library.

Symposium on Pulmonary Ventilation. Edited by R. P. Harbord, M.D. and R. Woolmer, M.D. Held in Leeds on February 19, 1958, under the auspices of The British Journal of Anaesthesia. Baltimore, Md., The Williams & Wilkins Co., exclusive U. S. agents. Cloth. 109 pages, illustrated, 1959. $4.00.

This brief work is a collection of papers and comments of participants in a conference on Pulmonary Ventilation. Views of experts in the various specialties are presented.

The discussions deal with Lung Gas Sampling and Analysis during Anaesthesia; Observations on Sampling Expiratory Gases during Artificial Respiration; Problems of Pulmonary Ventilation in the Newborn; A Clinical Note on the Difficulties of Maintaining Adequate Pulmonary Ventilation during Hypothermia; Management of Pulmonary Ventilation in Emphysematous Subjects in the State of Carbon Dioxide Narcosis and Mechanisms of Airway Obstruction.

The subject is of vital interest to anesthetists and will appeal to many readers in the field.

Principles and Practice of Obstetric Anaesthesia. By J. Selwyn Crawford, M.B., CH.B., D.A. (England), F.F.A.R.C.S., Anaesthesia Service, The Presbyterian Hospital, New York; Late Senior Registrar and First Assistant, Department of Anaesthesia, Central Middlesex Hospital, London. Springfield, Ill., Charles C Thomas. Cloth. 128 pages, 1959. $4.00.

This small volume presents the methods of obstetrical analgesia and anesthesia that are used in Great Britain. A review of the physiology of the pregnant woman, placental transmission, analgesia in labor, anesthesia for delivery, physiology of the newborn and equipment for the delivery room are subjects that will be of interest to anesthetists.

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(Continued from page 269)

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INDEX TO ADVERTISERS

Abbott Laboratories 232, 233, 239
Ayerst Laboratories 234
Burgess Publishing Co. 286
Burroughs Welcome & Co. 235
Ciba Pharmaceutical Products, Inc. 238
Foregger Co. 241
Linde Co., A Division of Union Carbide Corp. ..... IBC
Maginnis and Associates 289
Mallinckrodt 242
C. V. Mosby Co. 230
Ohio Chemical & Surgical Equipment Co. 231, 296
Parke, Davis & Co. 240
Pharmaseal Laboratories 229
Puritan Compressed Gas Corp. 236, 237
H. E. Richards Company, Inc. 280, 284
Union Carbide Corp., Linde Co. ..... IBC
Classified Advertisements 291

Journal American Association of Nurse Anesthetists Volume XXVII 1959 Index of Subject and Author

A

Aberg, Harriet L.: Safety in anesthetizing locations, 56, 132, 214, 283

Adrenal cortex insufficiency, 124

Adriani, John: Spinal anesthesia: complications and contraindications, part I, 11; spinal anesthesia: postanesthetic complications, part II, 115

American Association of Nurse Anesthetists: Annual meeting, announcement, 79, 228; program, 150; call to convention, 156; candidates for office, 205; officers, IFC August, IFC November

Amerson, J. Richard: Anesthesia in severe mediastinitis, 190

Aneleridine, use of (abstract), 218

Anesthesia: adrenal cortex in, 124; for bronchoscopy and bronchography, 176; carbon dioxide problems in, 264; for cardiac surgery, 107; dilemma in, 18; for esophageal resection, 256; for geriatrics, 91, 163, 193; effect of mediastinitis on, 190; in myasthenia gravis, 243; obstetrics, 28; for opthalmology (abstract), 217; in pediatrics, 270; spinal, 11, 115

Allen, J. Garrett: The dilemma in anesthesia: an analysis of current and future needs, 18

B

Barbiturate, bemegride antagonism (abstract), 66, 219

Beer, George E.: Geriatric anesthesia, 91

Bemegride, barbiturate antagonism (abstract), 66, 219

Berger, Olive L.: The use of respirators in the immediate postoperative period, 182

Bohne, Mildred T.: Is obstetrical anesthesia different?, 28

Book reviews: Anatomy and Physiology for Nurses, 71; Anatomy and Physiology Laboratory Manual, 134; Cyclopropane Anesthesia, 135; Eye Ear Nose and Throat Manual for Nurses, 211; Fundamentals of General Anesthesia for Students and Practitioners of Dentistry, 134; Handbook of Cardiology for Nurses, 72; Introduction to Human Anatomy, 211; A Manual of Anaesthetic Techniques, 211; Modern Trends in Anesthesia, 72; Orthopedic Nursing Procedures, 211; Physiology of Spinal Anesthesia, 134; Practical Blood Transfusion, 71; Principles and Practice of Obstetrical Anaesthesia, 290; The Recovery Room. A Symposium, 71; Resuscitation of the Unconscious Victim, 290; Symposium on Pulmonary Ventilation, 290

Boone, George D.: Anesthesia for bronchoscopy and bronchography, 176

Boone, Jeanette S.: Anesthesia for bronchoscopy and bronchography, 176

Bronchoscopy and bronchography, anesthesia for, 176

Buthalitone sodium (abstract), 67

C

Carbon dioxide, in anesthesia, 264

Chloralose-urethane, effect on heart (abstract), 286

Cleverdon, Jack: Obstructive pulmonary emphysema, 199

Complications, post spinal, 11, 115

Compton, Jessie L.: The role of the nurse anesthetist in a hospital safety program, 48

D

Dactil, see piperidolate (abstract), 136

Dental engines, dangers, 283

Dentistry, anesthesia for (abstract), 68

Drip dry cotten, uniforms of, 214

Drugs: newer hypnotics (abstract), 68

E

Electric plugs, hazards, 56

Elson, Matthew W.: Radiation hazards in diagnostic radiology, 275

Emphysema, obstructive pulmonary, 199

Endoscopy, use of meperidine in (abstract), 65

Esophageal resection, anesthesia for, 256

Explosions, hazards in operating room (abstract), 69

F

Fire, hazards (abstract), 69

Fluids, parenteral, safeguards (abstract), 136

Fluothane, action of (abstract), 137

G

Geister, Janet M.: We must walk together, 119

Geriatrics: anesthesia for, 91; anesthesia and surgery, 193; psychological and physiological considerations, 163

Gillesby, William J.: Esophageal resection in relation to anesthesia, 256

Greisheimer, Esther M.: Psychological and physiological considerations of the geriatric patient in relation to anesthesia, 163

H

Hayt, Emanuel, 58, 128, 215, 281

Heart: effect of chloralose-urethane (abstract), 286; effect of mephentermine (abstract), 285

Holzgrafe, Robert E.: Carbon dioxide problems in anesthesia, 264

Hospital safety, 56, 132, 214, 283; role of nurse anesthetist in, 48

Hypnotics, newer (abstract), 68

I

Insurance, 62, 140, 212, 288

L

LaBerge, Betty: Stress, adrenal cortical insufficiency, anesthesia and hydrocortisone, 124

Legislation: Broken needle during injection of tetanus antitoxin is not malpractice, 60; court holds expert testimony is not required to prove leaving foreign substance in wound is negligence, 129; death caused by spinal anesthetic is not by "accidental means" under double indemnity provisions of policy, 129; doctor liable for failure to disclose broken catheter left in patient's body, 281; professional liability (abstract), 67; malpractice case arising from injection of sodium pentothal should have been submitted to jury, 281; negligence of hospital nurses held cause of prolonged bleeding of patient in childbirth, 215; no cause of action for patient's fall from hospital bed without proof of need for side rails, 128; no liability in Connecticut for failure of nurses to use bedrails, 215; patient fails to prove that air embolism in intravenous feeding caused brain lesion, 59; surgeon guilty of malpractice in unwarranted removal of female organs, 58; surgeon required to explain presence of forceps in patient's abdomen after surgery, 58; unnecessary breast amputation, without pathological examination for cancer, held not negligence, 128

Levallorphan, effect on levorphanol (abstract), 138

Levorphanol, effect on levallorphan (abstract), 138

Lorfan, use in dentistry (abstract), 68

M

McBurney, Robert P.: Anesthesia and Surgery for the elderly patient, 193

McDonough, N. Pauline: The role of the nurse anesthetist in cardiac surgery, 107

Mediastinitis, anesthesia in, 190

Meperidine, in obstetrics (abstract), 67; in peroral endoscopies (abstract), 65

Mephentermine, effect on arrhythmias (abstract), 285

Meredith, Cameron W.: Psychology and the nurse anesthetist: a follow-up of actual experiences, 36

Morphine derivatives, new (abstract), 285

Murexine, action of (abstract), 66

Myasthenia gravis, anesthesia for, 243

N

Narbuth, Benjamin L.: A syringe holder for the anesthetist (note), 279

N.F.P.A., association report, 283

Neraval (abstract), 220

Newton, Saint Elmo: Anesthesia and surgery for the elderly patient, 193

Nisentil, use in dentistry (abstract), 68

Normorphine, potency of (abstract), 218

Nurse Anesthetists: professional status, 119; psychology, 36; role in cardiac surgery, 107; role in hospital safety program, 48

Nylon clothes, 56

O

Obstetrics: anesthesia, 28; anesthesia for (abstract), 67; promethazine in (abstract), 217

Operating Rooms: fire and explosion hazards in (abstract), 69; safety practices in, 132

Opthalmology, use of primicaine in (abstract), 217

P

Pediatrics, anesthesia problems in, 270

Physiology, of geriatric patient, 163

Piperidolate, antifibrillatory action in hypothermia (abstract), 136

Postoperative Care: use of normorphine (abstract), 218; use of respirators, 182

Primicaine, in opthalmic surgery (abstract), 217

Promazine, in obstetrics (abstract), 67

Promethazine, in surgery and obstetrics (abstract), 217

Psychology: 36, of geriatric patient, 163

Pulmonary edema, reflexes in (abstract), 65

R

Radiation, hazards in diagnostic radiology, 275

Radiology, radiation hazards in, 275

Respirators, postoperative, 182

S

Spinal: complications and contraindications, 11; postanesthetic complications, 115; use in obstetrics (abstract), 67

Stress, during anesthesia, 124

Suxamethonium, sensitivity to (abstract), 218

Syringe holder (note), 279

V

Vanderveen, James L.: Anesthesia for patients with myasthenia gravis, 243

Viadril, effect on heart (abstract), 286

W

Wrenn, Earle L.: Anesthesia problems in pediatric surgery, 270

X

X-ray, hazards, 275

Xylopropamine, analgesic properties of (abstract), 138

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